VOL. 52 NOVEMBER, 1956 No. 5

AMERICAN HEART JOURNAL

AN INTERNATIONAL PUBLICATION FOR THE STUDY OF THE CIRCULATION

Contents on Inside Cover

American Heart Journal

CONTENTS FOR NOVEMBER, 1956

Original Communications

Page

Displacement, Velocity, and Acceleration Ballistocardiograms as Registered With an Undamped Bed of Ultralow Natural Frequency. II. Instrumental Considerations. Maurice B. Rappaport, E. E., Boston, Mass. 643

Physical Basis of Ballistocardiography. IV. The Relative Movement of Subject and Ballistocardiograph. H. C. Burger, D.Sc., A. Noordergraaf, M.Sc., J. J. M. Korsten, M.Sc., and P. Ullersma, B.Sc., Utrecht, Netherlands 653

Auricular Escape During Vagus Stimulation After Crushing Sinus Node Tissue. D. Scherf, M.D., S. Blumenfeld, M.D., E. Reid, and A. Weisz, M.D., New York, N. Y. 674

Studies of Pulmonary Hypertension. VII. Hemodynamic Effects of Acute Hypoxia in Patients With Mitral Stenosis. Paul N. Yu, M.D., David C. Beatty, M.D., M.R.C.P., Frank W. Lovejoy, Jr., M.D., Robert E. Nye, Jr., M.D., and Howard A. Joos, M.D., Rochester, N. Y. With the Technical Assistance of Ruth Knight and Carol Gouverneur 683

Cardiac Metabolism in Experimental Ventricular Fibrillation. A. Pedersen, M.D., A. Siegel, M.S., and R. J. Bing, M.D., Birmingham, Ala. 695

A New Look at Electrocardiographic Leads. Abraham I. Schaffer, M.D., Serge Blumenfeld, M.D., Robert Bussan, M.D., and David Scherf, M.D., New York, N. Y. 704

A Study of Electrocardiographic and Spatial Vectorcardiographic Changes in Bundle Branch Block by Means of the Reconstruction Method. Hideo Toyoshima, Yasushi Mizuno, Mitsuo Hattori, and Yoshiko Saruhashi, Nagoya, Japan 711

The Ventricular Patterns in the Right Precordial Leads in Mitral Stenosis. Thomas L. Morris, M.B., B.Ch., and William Whitaker, M.D., M.R.C.P., Sheffield, England 738

The Diagnostic Significance of Tall Upright T Waves in the Chest Leads. J. Freundlich, M.D., Vancouver, British Columbia 749

The Effect of Boundary Contour on the Distribution of Dipole Potential in a Volume Conductor. Robert A. Helm, M.D., Cincinnati, Ohio 768

The Effect of Background Noise on Cardiac Auscultation. Dale Groom, M.D., Charleston, S. C. With the Technical Assistance of Oren Herring, B.S., Wofford Francis, and Gibson Shealy, B.S. 781

Clinical Reports

Pulmonic Valvular Regurgitation During Twenty-Seven Years After Gonorrheal Endocarditis. Knud Henning Olesen, M.D., and Jorgen Fabricius, M.D., Copenhagen, Denmark 791

Transient Ventricular Tachycardia Following the Valsalva Maneuver in a Patient With Paroxysmal Atrial Tachycardia. William Hollander, M.D., and George Entwisle, M.D., Boston, Mass. 799

Book Reviews

Book Reviews 804

Announcements

Announcements 806

(Editorial and Business Communications on Page 2 of Advertising Section)

Vol. 52, No. 5, November, 1956, American Heart Journal is published monthly, by The C. V. Mosby Company, 3207 Washington Boulevard, St. Louis 3, Missouri, entered as second-class matter January 23, 1917 at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Additional entry authorized at Jefferson City, Missouri. Subscription rates: United States and Its Possessions $13.00, Students $6.50; Canada, Latin-America, and Spain $13.50, Students $7.00; Other Countries $14.50, Students $8.00. Single copies, $2.00 postpaid. Printed in the U. S. A.

American Heart Journal

VOL. 52 NOVEMBER, 1956 NO. 5

Original Communications

DISPLACEMENT, VELOCITY, AND ACCELERATION BALLISTOCARDIOGRAMS AS REGISTERED WITH AN UNDAMPED BED OF ULTRALOW NATURAL FREQUENCY

II. INSTRUMENTAL CONSIDERATIONS

MAURICE B. RAPPAPORT, E.E.
BOSTON, MASS.

MECHANICAL DETAILS

THE simplest way to construct a bed with a natural frequency of 0.3 cycles per second or lower is to suspend it from the ceiling by way of four wires. When a bed is suspended in this manner, its performance is comparable to that of a pendulum which is generally suspended by means of a single wire or a pivoted rod and the formulae which apply to the pendulum are applicable. The parameters which control the natural frequency of a pendulum or suspended bed are expressed by the formula

$$f = \frac{1}{2\pi\sqrt{\frac{l}{g}}} \qquad (1)$$

where f = natural frequency in cycles per second,
l = length of suspension in feet, and
g = gravitational acceleration = 32.16 ft. per second per second at sea level.

From the formula (1) it is apparent the only parameters which control the natural frequency are suspension length and gravitational acceleration. The weight of the bed and the weight of the subject have no effect upon the natural frequency of the ballistocardiograph. In Table I are calculated natural frequencies which would result for different suspension lengths. In order to satisfy the minimal requirement where the bed would have a natural frequency of 0.3 cycles per second,[3] the bed suspension must be at least 9 ft. long. In order to attain an appreciable further diminution in natural frequency the suspension length must be increased appreciably; a natural frequency of 0.1 cycles per second requires a suspension length of 81 ft.

From the Ballistocardiographic Laboratory, Cardiology Department, Massachusetts General Hospital, Boston, Mass.
Read in part at the Second World Congress of Cardiology.[1]
Received for publication Aug. 2, 1955.

Henderson[2] suggested a procedure for attaining rather low natural frequencies without abnormal suspension lengths. He observed that if the bed were displaced laterally by means of two pivoted pins, the natural frequency of the

TABLE I. CALCULATED NATURAL FREQUENCY OF A SUSPENDED BED FOR DIFFERENT SUSPENSION LENGTHS

SUSPENSION LENGTH IN FT.	NATURAL FREQUENCY IN CYCLES PER SEC.
3	$\frac{1}{2\pi\sqrt{\frac{3}{32}}} = 0.52$
6	$\frac{1}{2\pi\sqrt{\frac{6}{32}}} = 0.39$
8	$\frac{1}{2\pi\sqrt{\frac{8}{32}}} = 0.32$
9	$\frac{1}{2\pi\sqrt{\frac{9}{32}}} = 0.30$
10	$\frac{1}{2\pi\sqrt{\frac{10}{32}}} = 0.28$
12	$\frac{1}{2\pi\sqrt{\frac{12}{32}}} = 0.26$
14	$\frac{1}{2\pi\sqrt{\frac{14}{32}}} = 0.25$
81	$\frac{1}{2\pi\sqrt{\frac{81}{32}}} = 0.10$

bed was lower than when hanging normally. The lateral bed displacement by means of the pivoted pins apparently produces a metronome effect. My experiments confirm Henderson's observations and, with a 6.5 ft. suspension which normally has a natural frequency of 0.35 cycles per second, it was a simple matter to obtain a natural frequency of 0.25 cycles per second or lower. The lowest natural frequency attainable was 0.1 cycles per second, and below this value the system became unstable in that the bed tended to fall to one side and could not pass the apex of the metronome or lateral arc.

Using the Henderson principle, a ballistocardiograph was constructed with a 6.5 ft. suspension which could duplicate the performance of an 81 ft. suspension. Ballistocardiograms were registered with a natural frequency of 0.25 and 0.1 cycles per second and no clinically measureable differences were observed. At natural frequencies above 0.3 cycles per second distinct temporal or phase distortion was observable as was theoretically predicted.[3]

When a bed is suspended from the ceiling it is most important to limit the bed motion to 1 degree of freedom in the head-foot axis. It would appear that if the bed were allowed to have 2 degrees of freedom such as in the head-foot and in the lateral directions, and if transducers were used for sensing the head-foot and lateral movements of the bed simultaneously, more pertinent ballistocardiographic information would be available. This is not so because the ballistic forces which are projected laterally would induce a rotary motion to the

Fig. 1.—Photograph of an experimental ultralow natural frequency ballistocardiograph.

bed. Furthermore, the exact location of the lateral transducer with respect to the body is a rather moot question. When the bed possesses a single degree of freedom, the rotary motion of the bed cannot occur and the true head-foot projections of the spatial vector minus transmissional losses in the body[3] are registered during the cardiac cycle. The double pivoted pin arrangement not only serves as a device for lowering the natural frequency of the system but as a mechanism for limiting the bed motion to a single degree of freedom.

Fig. 1 is a photograph of an experimental ultralow natural frequency ballistocardiograph such as has been used in my experiments. The original data were obtained from a ceiling-suspended bed with 12.5 foot suspensions. The metronome effect was employed to vary the natural frequency between 0.1 and 0.25 cycles per second. All details other than suspension length were retained in the later model, shown in Fig. 1, which had a suspension length of 6.5 ft.

If an apparatus such as the one in Fig. 1 is used, the degree of rigidity of the supporting frame is extremely critical; a nonrigid supporting frame will introduce artefacts in the recorded ballistocardiogram. From this consideration alone, it is advisable, if possible, to use a ceiling-suspended system and thus circumvent many constructional problems.

The bed frame is constructed of 61S-T1 aluminum which is a hard aluminum alloy. The outside diameter of the tubing is 1.5 inches with a wall thickness of 0.035 inch. The side and end tubes are joined by milling out one of the tubes with a 1.5 inch mill so that one tube will butt into the other. The two tubes at the butted joint are then clamped together with a bolt and rounded washers. The length of the bed is 72 inches and the width is 22.5 inches. At the foot end where the suspension wires are anchored, a cross brace 17 inches from the end is butt mounted on the underside of the bed. A cross brace is not placed at the head end as the subject would rest upon it with the normal give of the fabric. The rigidity of the bed has been found adequate without the latter brace.

Grade A airplane fabric United States Government Specification Number MIL-C-5646 and Civil Aeronautics Association Number AMS-3806 was used on the bed frame as shown in Fig. 1. All seams were reinforced with reinforcing tape such as United States Government Number MIL-T-5661, or equivalent, to prevent tearing. Double machine stitching was used throughout. The fabric form after fabrication was slipped onto the framework so that it was taut. At first, several coats of airplane dope were sprayed on the fabric, which shrunk the fabric and produced a drumlike effect. It was observed that the drumlike support had a tendency to produce a vertical bounce as a result of cardiac activity which introduced a small amount of artefact, whereas the softer undoped fabric did not introduce artefact. The total weight of the bed was 4 pounds.

The four bed-suspension wires were suspended from the top of the supporting framework to the bed to form two parallel isosceles triangles. The effective length of the suspension is equal to the length of the vertical bisecting line of an isosceles triangle. The wires were stranded steel cable with a nylon jacket. The jacket serves as a protective coating against corrosion and reduces secondary wire oscillations.

The lateral deflecting pins which may be seen in Fig. 1 located to the rear at the level of the bed are 4 inches long. The hardened tapered pin tips are set into tapered sockets located at the ends of threaded rods. The threaded rods are in turn run through threaded holes, and rotation of the rods moves the deflecting pins laterally for optimal adjustment of the metronome effect.

Mechanical stops were found desirable to limit the head-foot movement and a clamping device to steady the bed was used when the subject was placed on the bed or removed.

An ultralow natural frequency bed such as has just been described exhibits extremely low damping. There are many ways of producing slight damping (20 per cent of critical or less), such as frictional resistance, cotton pads, viscous fluid, etc. Very soft cotton pads have generally proved adequate although temperature-insensitive viscous fluid procedures lend themselves for more precise control. The most convenient procedure is gradually to increase damping until spontaneous bed sway does not occur—experience has shown that at this point the degree of damping is not in excess of 20 per cent of critical.

ELECTRICAL DETAILS

In selecting a transducer which is capable of sensing the ballistic movements of the suspended bed, a choice between magnetic, photoelectric, capacitance, strain gauge, piezoelectric, and differential transformer type transducers had to be made. For maximal convenience, the type of transducer should be compatible for operation with present day electronic electrocardiographs. This immediately eliminated the capacitance, strain gauge, and differential transformer type transducers, as special electronic components would have to be interposed between the sensing unit and the electrocardiograph.

Another desirable transducer characteristic is that it be self-generating to avoid the use of batteries, power supplies, optical systems, etc.; this requirement eliminated the photoelectric transducer. Additional desirable characteristics are fixed sensitivity during the life of the instrument and adequate sensitivity without additional amplification when displacement, velocity, and acceleration type ballistocardiographic recordings are to be registered. The piezoelectric transducer was found inadequate, as it did not satisfy the latter requirements. The final and probably the most important thing is that the transducer must not impede the free movement of the bed. The magnetic type transducer seemed to satisfy all the requirements listed but not in the customary form used in ballistocardiography.

A desirable form of magnetic transducer whose parameters are adequately flexible for ballistocardiographic applications is the bar-magnet velocity meter, described by Perls and Buchmann,[4] for the measurement of vibration and shock motions in physical applications. Later, Smith and Bryan[5] employed the bar-magnet velocity meter as the sensing element in a shin type ballistocardiograph. Basically the bar-magnet velocity meter consists of a long bar magnet and a long coil of wire. When one end of the magnet is placed halfway into the coil and a relative movement between magnet and coil occurs, a voltage is generated in the coil. The generated voltage is dependent upon the rate at which the magnetic lines of force are cut by the coil, and thus a velocity measuring device results. When used on the ultralow natural frequency bed, the bar magnet, which weighs 9 ounces, is attached to the bed and the coil is fastened to the frame (Fig. 1) or to a rigid support if the bed is suspended from the ceiling. The relative movements between the fixed coil and the moving bed are thus sensed. This seems like an extremely simple arrangement but there is considerably more involved than appears at first glance. Actually, many design parameters must be taken into consideration, such as magnet position relative to the coil versus

output voltage, sensitivity, and linear range. Measurements show that an alnico 5 magnet with a flux density reduced from saturation by 5 per cent or more is adequately stable over an indefinite period of time. The optimal magnet length was found to be 4.75 inches with a diameter of 0.75 inch. The optimal coil was 3 inches long with an outside diameter of about 2 inches and an inside diameter of 1 inch. The coil was made up of 40,000 turns of number 38 enamel insulated copper wire, layer wound, with an electrical resistance of approximately 10,000 ohms.

In order to determine the sensitivity of the transducer as a velocity measuring device in addition to determining its linear range, the following experiment was performed. The coil was set in a vertical position and the magnet was allowed to move through the coil at a known fixed rate. A convenient way to

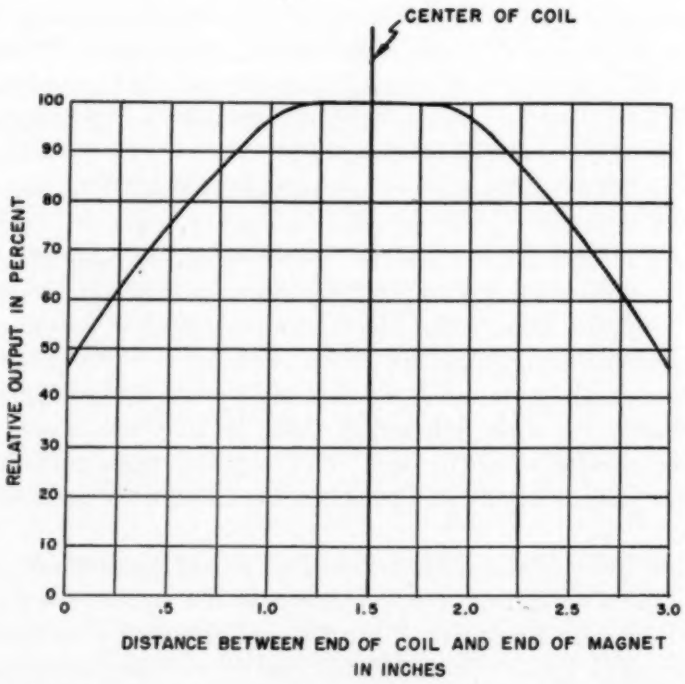


Fig. 2.—Velocity calibration curve of the ballistocardiograph sensing transducer.

control the magnet movement precisely was by attaching a string to one end of the magnet with cement and the other end of the string to the recording paper of a Sanborn Viso-Cardiette. The paper or chart speed of an electrocardiograph has a linear speed of 25 mm. per second, which is held to precise tolerances. Pulleys were used to direct the movement of the magnet relative to the coil.

The relative movement between the magnet and coil generated a voltage in the coil whose magnitude depended upon the velocity at which the magnet moved. The generated voltage was in turn fed into a Sanborn Twin-Viso direct coupled amplifier which was calibrated to a sensitivity of 50 millivolts per centimeter deflection. The resultant curve which was registered by the Twin-Viso during the passage of the magnet through the coil is shown in Fig. 2. The input of the D.C. Amplifier is of the order of 5 megohms; therefore we may consider the measured transducer voltages as open circuit voltages. From Fig. 2 it is

apparent that a linear working range exists throughout the middle half inch of the transducer coil with a sensitivity equal to 4.5 volts per inch per second.

Ballistic body movements may be measured in terms of magnitude (displacement), body speed (velocity), and the acceleration which the body experiences in attaining the speed of traversal.* Mathematically, body motion may be represented as a vector which possesses magnitude, direction, and sense; velocity is the derivative* of displacement, and acceleration is the derivative of velocity. Expressed mathematically for a sinusoidal excitation,

$$d = D \sin \omega t \tag{2}$$

$$v = D\omega \cos \omega t \tag{3}$$

$$a = -D\omega^2 \sin \omega t \tag{4}$$

where d = instantaneous displacement,
v = instantaneous velocity,
a = instantaneous acceleration,
D = maximum instantaneous displacement,
ω = angular frequency in radians per second, and
t = time.

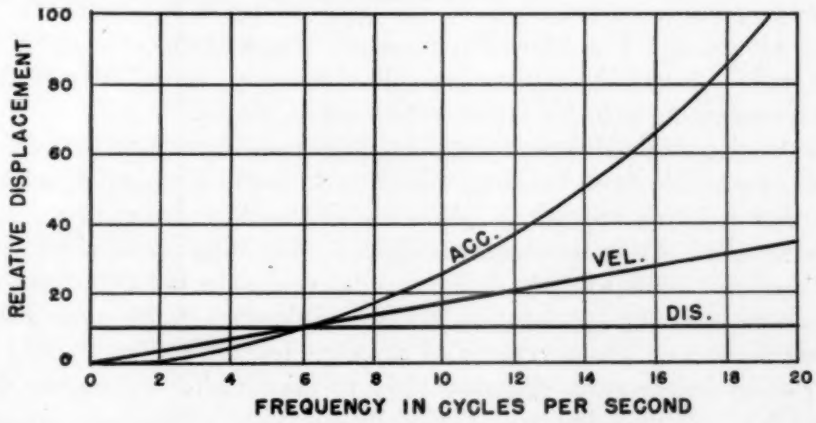


Fig. 3.—Frequency response curves which show the relationship of displacement, velocity, and acceleration to a sinusoidal stimulus of constant magnitude.

Frequency response curves which show the relationship of displacement, velocity, and acceleration to a sinusoidal stimulus of constant magnitude are shown in Fig. 3. The displacement response is constant for an applied force no matter what its sinusoidal frequency of vibration may be as long as the magnitude of the force is unchanged. The velocity response increases in magnitude to the constantly applied sinusoidal stimulus as the frequency is increased. The rate of increase is a doubling in magnitude of deflection as the frequency is doubled. The acceleration response curve shows a fourfold increase in magnitude of deflection as the frequency is doubled.

*When an object traverses a distance at a variable speed, differential calculus allows us to establish, at any instant of time during the traversal, the instantaneous velocity and acceleration. For example, the velocity at any instant is the instantaneous distance change divided by the instantaneous time change which is the first derivative of the distance change with respect to time change; the instantaneous acceleration change is the second derivative.

The magnitude of the voltage generated in the coil may be expressed by the formula

$$e = 10^{-8} N \frac{d\phi}{dt} \text{ volts} \quad (5)$$

where e = voltage generated in the coil as a result of relative movement between magnet and coil,
N = number of turns of wire in the coil,
ϕ = flux linkages or magnetic lines of force which surround the coil, and
t = time.

According to formula (5), the voltage is equal to the product of the number of turns in the coil by the rate at which the lines of force are cut. That is, as a force of higher frequency is applied, the rate at which the lines of force are cut is increased, and, therefore, the voltage is higher even though the magnitude of relative movement between coil and magnet is unchanged; the rate of rise is such as to correspond to the velocity of relative movement of magnet and coil. In order to attain a displacement response, all that is necessary is to integrate the velocity response as indicated by the relationship of equations (2) and (3). Likewise, to attain an acceleration response the transducer output must be differentiated. Electrical circuitry capable of integrating and differentiating the voltages representing velocity are incorporated in Fig. 4.

When registering ballistocardiograms, it is most important that the location be as free as possible from building vibration to avoid a ragged baseline. It so happens that building vibrations fall in about the same frequency spectrum as those encountered in cardiovascular ballistics. The displacement ballistocardiogram is the least susceptible to building vibrations, the velocity ballistocardiogram more so, and the acceleration ballistocardiogram is the most susceptible. The rising frequency characteristic of velocity response (Fig. 3) and the still greater rise in acceleration response tend to accentuate the higher frequency building vibrations as indicated. Generally speaking, a basement room is most free of vibration with proportional increase as one goes to the upper floors.

The use of low-pass filters for the attenuation of building vibration has proved to be of limited value. Ballistocardiograms as registered with an undamped bed of ultralow natural frequency has frequency components at least up to 40 cycles per second. If a low-pass filter is used with a cutoff somewhat above this upper frequency portion of the ballistic spectrum, the artefacts due to building vibration are suppressed very little.

With the circuitry shown in Fig. 4, it is possible simultaneously to register displacement, velocity, and acceleration ballistocardiograms if a multichannel electrocardiograph is available. If for example a four-channel electrocardiograph is used, another cardiovascular event may be registered in the fourth channel for orientation and timing of ballistic events in the cardiac cycle; the most useful have been the electrocardiogram, the sphygmogram, the apex cardiogram, and the phonocardiogram.

The circuitry of Fig. 4 is arranged with suitable voltage dividers and differentiation and integration constants selected so that on the average subject the

displacement, velocity, and acceleration ballistocardiograms register with comparable magnitude when all channels are standardized equally. The velocity calibration is obtained as previously discussed but with the circuitry of Fig. 4 instead of with the open circuit technique previously discussed. Displacement calibration may be obtained by blocking the bed against a stop and inserting a thin strip of metal (0.005 inch) between the stop and the bed. The deflection of the recorder corresponds to the bed displacement which is equal to the thickness of the strip in the displacement calibration. Another way of arriving at the dis-

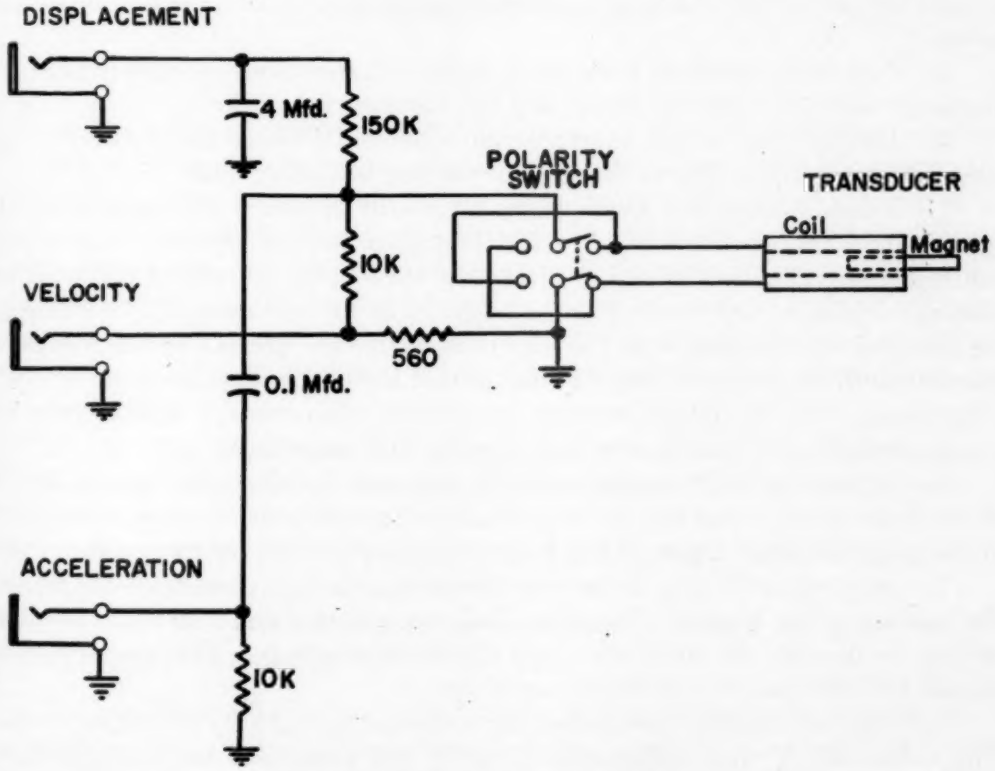


Fig. 4.—The electrical circuitry associated with the ballistocardiograph.

placement calibration is by calculation from the circuitry of Fig. 4; acceleration may be similarly evaluated. The calibration values used are

Displacement: 3.5 millivolts per 0.001 inch,
Velocity: 0.12 millivolts per 0.001 inch per second, and
Acceleration: 2.4 millivolts per inch per second per second.

In order to register ballistocardiograms, connection is made from the jacks marked "displacement," "velocity," and "acceleration" to three corresponding channels on a multichannel recorder. The frequency response characteristics of the ordinary electrocardiographic resistance-capacity coupled amplifier are adequate for ballistocardiography—direct coupled amplification is unnecessary as the lowest frequency that must be recorded accurately is the fundamental heart rate.

A reversing switch is shown in Fig. 4 for the purpose of obtaining correct polarity in the ballistocardiogram. That is, a positive deflection must represent a headward body movement.

CONCLUSIONS

1. When a subject is placed on an ultralow natural frequency bed of the pendulum type, the natural frequency of the bed is unaffected by the weight of the subject.

2. To attain appreciable diminution in natural frequency in an ultralow natural frequency bed, the length of the suspensions must be increased by a large factor.

3. A metronome effect, when used on the bed, effectively reduces the natural frequency without markedly increasing the suspension length.

4. Pivoted pins which produce the metronome effect also limit the bed movement to a single degree of freedom—in the head-foot axis.

5. When 2 degrees of freedom are used such as lateral and head-foot, the bed acquires a rotary motion as a result of cardiovascular activity. The lateral ballistocardiogram, if registered, is affected by the relative placement of the lateral sensing transducer with respect to the body. The further away the lateral sensing transducer is located from the axis of activity, the greater is the rotational moment and, in turn, the larger is the lateral ballistocardiographic error. The exact location of the lateral sensing transducer, with respect to the body for minimal effects of moment, is a very questionable procedure.

6. When the bed possesses a single degree of freedom, the rotary motion of the bed cannot occur and the true head-foot projections of the spatial vector minus transmissional losses in the body are registered during the cardiac cycle.

7. A bar-magnet type transducer can be designed to possess all the properties necessary for ultralow frequency ballistocardiography and with adequate output to operate an electronic type electrocardiograph. The constructional details and methods of calibration are given.

8. The bar-magnet type transducer measures the velocity of bed movement. The electrical circuitry which can integrate the potentials related to velocity for bed displacement registrations is described. In addition, the electrical circuitry suitable for differentiating the potentials related to velocity for acceleration registrations is described.

9. The calibration constants are given for the measurement of actual body displacement, velocity, and acceleration which result from the activity of the cardiovascular forces.

REFERENCES

1. Rappaport, M. B.: Displacement, Velocity and Acceleration Ballistocardiograms as Registered With an Undamped Bed of Ultra-Low Natural Frequency, Abstracts of papers 2nd World Congress of Cardiology and 27th Annual Scientific Sessions of Am. Heart Assoc., p. 229, 1954.
2. Henderson, V.: The Mass-Movements of the Circulation as Shown by a Recoil Curve, Am. J. Physiol. **14**:287, 1905.
3. Rappaport, M. B.: Displacement, Velocity, and Acceleration Ballistocardiograms as Registered With an Undamped Bed of Ultralow Natural Frequency. I. Theory and Dynamic Considerations, AM. HEART J. **52**:483, 1956.
4. Perls, T. A., and Buchmann, E.: The Bar Magnet Velocity Meter, Rev. Sci. Inst. **22**:275, 1951.
5. Smith, J. E.: A Calibrated Bar-Magnet Velocity Meter for Use in Ballistocardiography, AM. HEART J. **44**:872, 1952.

PHYSICAL BASIS OF BALLISTOCARDIOGRAPHY. IV

The Relative Movement of Subject and Ballistocardiograph

H. C. Burger, D.Sc., A. Noordergraaf, M.Sc., J. J. M. Korsten, M.Sc., and P. Ullersma, B.Sc.

Utrecht, Netherlands

INTRODUCTION

TO GATHER information about human and animal circulatory problems several types of ballistocardiographs are in use. By the periodic contraction of the heart, mass displaces within the body (only the longitudinal axis is attended). Assuming that the binding between the subject's body and the ballistocardiograph (bcg; the ballistocardiogram will be abbreviated as BCG) was infinitely strong and the subject himself or herself was a rigid body, it appeared from a preceding paper[1] that the following quantity concerning the common center of gravity of subject and bcg is measured if the *displacement* of the bcg is recorded:

A. The displacement of the common center of gravity of subject and bcg, using a *low-frequency* bcg, according to Gordon,[2] Henderson,[3] and Burger,[4]

B. The velocity of the center of gravity using a *middle-frequency* bcg, according to Nickerson,[5] and

C. The acceleration of the center of gravity, using a *high-frequency* bcg, according to Starr.[6]

The requirements that these bcg's have to meet follow from considerations discussed in one of the above-mentioned papers.[1] Moreover, it has been shown in that paper what are the mutual relations between the records procured by these types of bcg's, and in which way all three quantities, displacement, velocity, and acceleration, concerning the common center of gravity of subject and bcg, were to be found by any of the three above-mentioned types of bcg's. The last mentioned idea has been worked out and applied already for the low-frequency bcg by Elsbach[7] and, but partly, for the Dock type by Smith and Bryan.[8]

The force acting on the body follows from the recorded acceleration by multiplication of the moving mass of subject and bcg and its acceleration at every moment during a heart cycle.

However, in reality the binding between the subject's body and the bcg is not infinitely strong. Because of the tissue layer between the skeleton of the subject and the bcg there will be a difference in movement of subject and bcg.

From the Department of Medical Physics, Physical Laboratory of the University, Utrecht.
Received for publication Feb. 28, 1956.

To this phenomenon attention was drawn for the first time, as far as we know, in our paper concerning the physical basis of ballistocardiography,[4] in which this difference in movement, the "relative movement," was shown experimentally for the low-frequency bcg.

In the present paper the influence of this relative movement of subject and bcg will be investigated for the three mentioned types of bcg's and for the Dock type (direct-body type). Some results of this investigation were communicated in the discussion by the present authors of a paper by Nickerson.[9,10] The movement of the heart with respect to the skeleton will not be discussed in this paper.

THEORY

In its simplest form the problem of the relative movement of subject and bcg is a special form of a more general problem: that of two coupled harmonic oscillators.

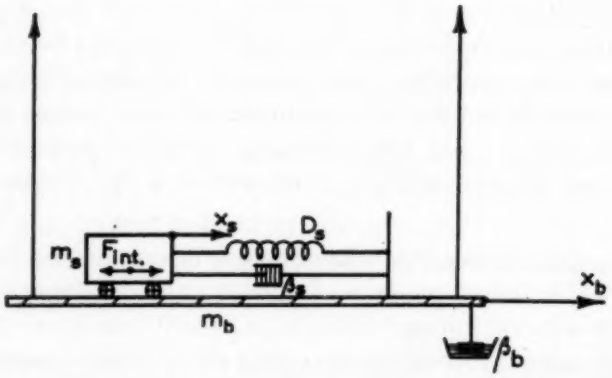


Fig. 1.—Schematical representation of the ballistocardiographic system in which the relative movement is taken into account. The subject with mass m_s is coupled to the bcg with mass m_b by a directive force and a frictional force. The whole system is coupled to the surroundings in an analogous way.

The problem in the form we have to face it can schematically be represented as in Fig. 1, as was done in a preceding paper.[9] The subject with mass m_s is represented by a cart that is coupled to the bcg with mass m_b by a directive force and a frictional force. The system of subject and bcg is coupled to the surroundings in the same way.

The internal force F_{int} equals $m_s\ddot{x}_c$, x_c, $\dot{x}_c$, and $\ddot{x}_c$, respectively, being the displacement, the velocity, and the acceleration of the center of gravity, calculated with respect to the skeleton and positive to the left in Fig. 1 (footward). The force exerted on the body equals this force but has an opposite direction (action equals reaction). The displacement, x_s, the velocity, $\dot{x}_s$, and the acceleration, $\ddot{x}_s$, of the subject are chosen positive to the right in Fig. 1 (headward). The same holds good for the respective quantities x_b, $\dot{x}_b$, and $\ddot{x}_b$ of the bcg. The frictional force acting between subject and bcg is assumed proportional to the difference in velocity of subject and bcg and acts in a direction opposite to this difference. So the frictional force exerted on the subject by the bcg can be written in the form $-\beta_s(\dot{x}_s - \dot{x}_b)$ and that exerted on the bcg by the subject $\beta_s(\dot{x}_s - \dot{x}_b)$. In an analogous way the directive force acting on the body can be written in the

form $-D_s\,(x_s - x_b)$; that exerted on the bcg by the body has again the opposite sign. D_s is the proportionality factor between the difference in displacement and the force. The total force acting on the subject equals the product of the mass m_s of the subject and its acceleration $\ddot{x}_s$, so:

$$m_s\ddot{x}_c - \beta_s\,(\dot{x}_s - \dot{x}_b) - D_s\,(x_s - x_b) = m_s\ddot{x}_s \tag{1a}$$

In the same way the differential equation for the movement of the bcg can be found. Besides the two forces $\beta_s\,(\dot{x}_s - \dot{x}_b)$ and $D_s\,(x_s - x_b)$ two other forces act on the bcg. They are the frictional force $-\beta_b\dot{x}_b$, caused by the applied damping of subject and bcg with respect to the surroundings and $-D_bx_b$, caused by the directive force acting between the bcg and the surroundings. The sum of these forces equals, once again, the product of the mass m_b of the bcg and its acceleration $\ddot{x}_b$, so:

$$-\beta_b\dot{x}_b - D_bx_b + \beta_s\,(\dot{x}_s - \dot{x}_b) + D_s\,(x_s - x_b) = m_b\ddot{x}_b \tag{2a}$$

In this way two simultaneous differential equations describing the movement of subject and bcg are found. They read as follows, written in a more usual form, to be found from the equations (1a) and (2a) by interchanging some terms:

$$m_s\ddot{x}_s + \beta_s\,(\dot{x}_s - \dot{x}_b) + D_s\,(x_s - x_b) = m_s\ddot{x}_c \tag{1}$$

$$m_b\ddot{x}_b + \beta_b\dot{x}_b + D_bx_b + \beta_s\,(\dot{x}_b - \dot{x}_s) + D_s\,(x_b - x_s) = 0 \tag{2}$$

To find the solution of these differential equations, we cannot entirely do without mathematics.

The center of gravity of the subject is assumed to move periodically and, therefore, its movement can be developed in a Fourier series. An arbitrary term of such a series can be written in the usual exponential form

$$x_c = |\,x_c\,|\,e^{j\omega t} \tag{3a}$$

in which x_c is the displacement and $|x_c|$ is the amplitude of this displacement of the center of gravity, $\omega = 2\pi\nu$, with ν the frequency, t the time, and j the imaginary unit.

The solution of the differential equations (1) and (2) (valid after a time until the movement of the bcg is stationary) is:

$$x_s = |\,x_s\,|\,e^{\,j(\omega t + \varphi_s)} \tag{3b}$$

$$x_b = |\,x_b\,|\,e^{\,j(\omega t + \varphi_b)} \tag{3c}$$

in which $|x_s|$ and $|x_b|$ represent the amplitudes of the displacement of subject and bcg, respectively. φ_s and φ_b represent the phase shift (time-lag) between the mass-movement within the subject and the movement of subject and bcg, respectively.

Substituting the formulas (3a), (3b), and (3c) in the differential equations (1) and (2), the amplitude distortion and the phase shift (see below) can be derived.

After some calculation we get:

a. for the amplitude of the subject:

$$|\,x_s\,| = m_s\,\omega^2 M_s\,|\,x_c\,| \tag{4}$$

b. for the amplitude of the bcg:

$$| x_b | = m_s \omega^2 M_b | x_c | \tag{5}$$

in which the abbreviations M_s and M_b stand for:

$$M_s = \left[\frac{(-m_b\omega^2+D_s+D_b)^2+\omega^2(\beta_s+\beta_b)^2}{\{m_s m_b\omega^4-\omega^2(m_sD_b+m_bD_s+m_sD_s+\beta_b\beta_s)+D_sD_b\}^2+\{-\omega^3(m_s\beta_s+m_s\beta_b+m_b\beta_s)+\omega(\beta_sD_b+\beta_bD_s)\}^2} \right]^{\frac{1}{2}} \tag{4a}$$

$$M_b = \left[\frac{D_s^2+\omega^2\beta_s^2}{\{m_s m_b\omega^4-\omega^2(m_sD_b+m_bD_s+m_sD_s+\beta_b\beta_s)+D_sD_b\}^2+\{-\omega^3(m_s\beta_s+m_s\beta_b+m_b\beta_s)+\omega(\beta_sD_b+\beta_bD_s)\}^2} \right]^{\frac{1}{2}} \tag{5a}$$

c. for the ratio between the amplitude of the relative movement and the amplitude of the movement of the subject:

$$\left| \frac{x_s - x_b}{x_s} \right| = \left[\frac{(m_b\omega^2 - D_b)^2 + \beta_b^2\omega^2}{(-m_b\omega^2 + D_s + D_b)^2 + \omega^2(\beta_s + \beta_b)^2} \right]^{\frac{1}{2}} \tag{6}$$

From this general solution follow the amplitude characteristics of the different types of bcg's.

A. As is referred to in the introduction, the displacement of the *low-frequency* bcg, if applied correctly, represents the displacement of the center of gravity.[1] The way in which the amplitude of this displacement of the center of gravity is distorted if the displacement of the subject is recorded is described by

$$|x_s| = m_s\omega^2 M_s |x_c| \tag{4}$$

and by

$$|x_b| = m_s\omega^2 M_b |x_c| \tag{5}$$

if the displacement of the bcg is recorded.

B. The displacement of the *middle-frequency* bcg represents, with a certain approximation, the velocity of the center of gravity.[1] The way in which the amplitude of this velocity is distorted can be deduced from formula (4) after a simple calculation presented earlier.[1] In the case where the displacement of the subject is recorded it follows that:

$$|x_s| = m_s\omega M_s |\dot{x}_c| , \tag{7}$$

and in the case that the displacement of the bcg is recorded:

$$|x_b| = m_s\omega M_b |\dot{x}_c| . \tag{8}$$

C1. The displacement of the *high-frequency* bcg according to Starr represents, with a certain approximation, the acceleration of the center of gravity.[1] The way in which the amplitude of this acceleration is distorted is to be found from formula (4). In the case that the displacement of the subject is recorded it follows that:

$$|x_s| = m_s M_s |\ddot{x}_c| , \tag{9}$$

and in the case that the displacement of the bcg is recorded:

$$|x_b| = m_s M_b |\ddot{x}_c| . \tag{10}$$

C2. Although the bcg, according to Dock,[11] does not fit this scheme, it can be discussed here. This bcg has a table that is assumed infinitely strongly bound to the surroundings. So the natural frequency of the (loaded) bcg is very high with respect to the frequency of the heart. Therefore, the Dock type bcg has to be considered as a *high-frequency* bcg. As there is only one degree of freedom

in movement (the amplitude of the bcg equals zero), the distortion can be found by direct calculation.[9] The same result can be found from the formulas (9) and (10), holding for this type of bcg, by substituting the infinitely great value of D_b or m_b in formula (4a). This substitution must be made because of the fact that the binding of the bcg is infinitely great. So when the displacement of the subject is recorded, it follows that:

$$|x_s| = m_s M_s' |\ddot{x}_c| , \tag{11}$$

in which

$$M_s' = \left[\frac{1}{(-m_s\omega^2 + D_s)^2 + \omega^2\beta_s^2} \right]^{\frac{1}{2}} \tag{12}$$

From the same substitution in formula (5a) it follows that $M_b' = 0$, so the amplitude of the bcg $|x_b|$ equals zero too, as can be seen without calculation.

In all cases, except that of the low-frequency bcg, the ratio between the amplitude of the relative movement and the amplitude of the movement of the subject $|x_s - x_b| / |x_s|$ is so great that it is superfluous to calculate this relation in those cases beside the calculation of the amplitude characteristics for $|x_s|$ and $|x_b|$.

In the case of the high-frequency bcg, according to Dock, the ratio $|x_s - x_b| / |x_s| = 1$, since $x_b = 0$.

The time-lag between the phenomena occurring within the body and the movement of the body is indicated by the phase shift φ_s in formula (3b), that between the occurrences within the body and the movement of the bcg by the phase shift φ_b in formula (3c).

The phase shifts φ_s and φ_b can, just as the amplitudes, be calculated from the differential equations (1) and (2) by substituting the formulas (3a), (3b), and (3c). From this calculation it follows, if the displacement of the subject is recorded, that:

$$tg\ \varphi_s = \frac{uv - tw}{tv + uw} , \tag{13}$$

if the displacement of the bcg is recorded:

$$tg\ \varphi_b = \frac{u'v - t'w}{t'v + u'w} , \tag{14}$$

in which

$$t = -m_b\omega^2 + D_s + D_b \qquad u = \omega(\beta_s + \beta_b)$$

$$t' = D_s \qquad u' = \omega\beta_s$$

$$v = m_s m_b \omega^4 - \omega^2(m_s D_b + m_b D_s + m_s D_s + \beta_b\beta_s) + D_b D_s$$

$$w = -\omega^3(m_s\beta_b + m_b\beta_s + m_s\beta_s) + \omega(\beta_s D_b + \beta_b D_s) .$$

The value of the phase shifts φ_s^* and φ_b^* if the velocity or the acceleration of subject or bcg is recorded, can be found from φ_s and φ_b with the relations derived earlier.[1]

Ballistocardiographs that have to give a reliable representation of the quantities one is interested in ought to have amplitude and phase characteristics meeting certain requirements. We have chosen for these requirements:

1. The amplitude characteristic must be flat within 10 per cent in the frequency range from 1 to 30 c/s.

2. The phase characteristic must show a phase shift less than 20 degrees (0.06 period) in the same frequency range.

We have chosen the frequency range up to 30 c/s, because in the acceleration records made in our group, we noticed details corresponding to frequencies up to that amount.

In some cases the first requirement is not met. Because of this fact it is not necessary to investigate the fulfilment of the second requirement in those cases.

With the above-derived formulas (4), (5), and (7) to (11) for the amplitude characteristics and with (13) and (14) for the phase characteristics alone, it is not possible to calculate amplitude and phase characteristics. Two of the constants that need to be known (m_s, m_b, β_s, β_b, D_s, and D_b), namely β_s and D_s, cannot be deduced from the experimental circumstances. We therefore measured them separately on several subjects.

EXPERIMENTS

The measurements of β_s and D_s were done with the subject lying on a bcg of the low-frequency type, but clamped rigidly, and next lying on a rigid underlayer (Dock type). The subject was always in a supine position.

Our low-frequency bcg consists of a rectangular frame in which a piece of canvas has been stretched. The frame is suspended on four wires of about 3 meters length. A further description is to be found in a previous paper.[4] To measure β_s and D_s the bcg was fixed to the surroundings. On the foot end of the bcg a footplate is attached to the frame. The subject lying on the bcg can exert a force F on this footplate by pressing his feet toward it. This force F exerted on the footplate causes a spring to be impressed. F can be measured from the impression of this spring. The force exerted on the footplate also acts on the feet.

By a tap on the shoulders the subject, lying on the bcg, is given a deflection from its zero position. Then the subject oscillates about the equilibrium position with decreasing amplitude. This oscillation is recorded in the following manner. A light screen is attached to one of the shins. When the subject is in zero position, the screen's edge is in about the middle of a lightbeam incident on a phototube. When the subject is oscillating, alternatively more and less light is transmitted to the phototube. Between the terminals of the resistance in the phototube circuit a voltage arises proportional to the amount of incident light. The variations of voltage can be recorded with the aid of an electrocardiograph or cathode-ray oscillograph. This phototube method of recording, described earlier by us,[4] has not been changed in principle, but in practical arrangement only. In the light beam two small mirrors are placed, so that the light beam changes direction twice over ninety degrees. In this way a considerable diminution of the volume of the box containing the light source, the lenses, the phototube (and the added cathode-follower) was made possible. The present dimensions are 30 by 13 by 8 cm.^{3}, so that the box is easily displaceable.

The oscillating movement of the body after giving a tap on the shoulders is recorded on two places of the body.

1. A screen of about 60 Gm. was attached to a shin in such a way that the screen pointed through a hole in the canvas to the floor. By placing the phototube-box on the floor under the shin the movement of the shin was recorded.

2. A screen of about 20 Gm. was attached to the head in such a way that the screen pointed in the direction of the axis of the body. By fixing the phototube-box to the frame of the bcg the movement of the head was recorded.

As the screens were very light and very firmly attached to the body, the natural frequencies in the different directions in which oscillation was possible were not lower than 70 c/s. Moreover, the damping of these oscillations was about critical. Because of these reasons, together with the consideration that we are only interested in frequencies less than 30 c/s, the screens will follow the movement of the leg and the head so exactly that the difference in movement between leg and corresponding screen and between head and corresponding screen is negligible.

Measurements were done on leg and head to find a possible important difference in movement of leg and head (i. e., in the respective β_s and D_s) caused by the fact that head, spine, and leg are more or less movable with respect to each other.

These two places were chosen since they are convenient for recording the BCG. As will be shown below the binding between head, spine, and legs is fairly strong for the longitudinal movement of the body. Then, recording the movement of a screen attached to a leg is preferable to that of a screen attached to the head because the subject's head is apt to make a rolling movement. To prevent the latter movement, our subjects were invited to lay their heads in a ⌣-shaped quilted wooden frame of 10 cm. length, measured along the subject's axis.

Using this kind of screen with a natural frequency comparatively high with respect to the frequencies one is interested in, it is no longer necessary to make further investigations into the influence of the screen on the movement of the body, or to calculate the distortion of the body movement caused by the relative movement of the transducer (screen), as is done by Smith and Rosenbaum[12] and Smith, Rosenbaum, and Ostrich.[13] In their case the natural frequency is not high enough and the mass of the transducer not small enough to prevent this complication.

The recordings of the free vibration of the subject on the fixed bcg were done under the following circumstances:

1. Without the use of a footplate ($F = 0$ Kg.), while the canvas in the frame of the bcg was not stretched.
2. With the use of a footplate exerting a force on the feet of 25.10^6 dynes ($F = 25$ Kg.). Canvas as under 1.
3. $F = 0$ Kg., while the canvas in the frame was stretched.
4. $F = 25$ Kg., canvas as under 2.

Moreover, measurements on the free vibration were done on a rigid underlayer (a plank fixed on a concrete floor) to procure data on the Dock type. The previously mentioned values of F were used. The shins of the subjects lay across a piece of wood with a height of about 10 cm., according to Dock's indication.

After a few experiments we stopped using shoulder clamps combined with a footplate to make the binding between body and bcg as large as possible, as is done by von Wittern.[14] Only the footplate was used for this purpose. In our opinion, the shoulder clamps were too troublesome to the subject for routine use.

It is not possible to improve the fixation of the bcg to the subject by increasing the force F, exerted by the footplate on the feet above the value of 25.10^6 dynes (25 Kg.). With larger F the subject will glide over the canvas or the plank. On the other hand, not every arbitrarily chosen spring that can exert a force of 25 Kg. is useful for this purpose. For a better coupling of subject and bcg an increase of the directive force per centimeter D_s is required of the subject's tissue layer between the skeleton and the bcg, and an increase of the damping coefficient β_s. One should prefer to choose the directive force per centimeter D_f of the footplate large with respect to D_s. However, this is not possible without making use of shoulder clamps. Therefore a spring must be chosen that impresses a few centimeters to exert a force of 25 Kg., to prevent a little gliding movement of the subject from causing the loss of contact between feet and footplate. But a spring with D_f large with respect to D_s, impressed a few centimeters, exerts a force on the feet much greater than 25 Kg. and causes gliding. So a spring must be chosen with the property that D_f is smaller than D_s.

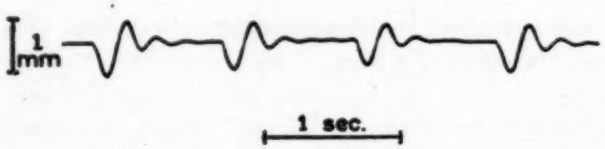


Fig. 2.—Damped vibrations of the subject lying on a rigid underlayer, caused by taps on the shoulders.

A tap on the shoulders generating the free vibration must be so great that the displacement caused by the heart action has an amplitude that is small with respect to the amplitude of the free vibration. On the other hand, the amplitude of the vibration must be small enough to provide proportionality. As a compromise we have chosen a beginning amplitude of about 0.5 mm. Fig. 2 represents a record of four successive taps, the subject lying on a rigid underlayer.

CALCULATIONS AND RESULTS

The records of the free vibrations must be conceived as a superposition of the vibrations of two coupled systems: the head on one hand and trunk and legs on the other. So, the record of the movement of a shin is a superposition of two vibrations with different amplitudes and different frequencies. In general, the movement of the head will be another superposition of the same frequencies.

The alinear properties of the tissue layer between skeleton and bcg may be a second reason why the ratio of the amplitudes in two consecutive inversion points and the vibration time are not constant during one vibration.

As the coupling between the above-mentioned systems is great and the amplitude of the vibration is small we assumed the vibration to be that of one degree of freedom. The values of β_s and D_s can then be calculated by making use of the following formulas[4]:

$$\beta_s = \frac{4\, m_s\, ln\, x_1/x_2}{T} \tag{15}$$

$$D_s = \frac{16\,\pi^2\, m_s^2 + \beta_s^2\, T^2}{4\, m_s\, T^2} \tag{16}$$

T is the time of oscillation of the free vibration and x_1/x_2 is the ratio of the amplitudes in two consecutive inversion points (Fig. 2). From our records we calculated mean values of β_s and D_s during a vibration caused by one tap and during a few oscillations caused by several taps.

The values of β_s and D_s were measured on subjects of various age groups. The numerical values of β_s and ν_s ($D_s = 4\pi^2 \nu_s^2 m_s$) are listed in Tables I, II, and III. (The natural frequency if there was no damping is called ν_s.) The measurements listed in Table I were done with a screen attached to a shin, while the subjects lay on a nonstretched canvas. In the columns indicated with I in Table II, the canvas was not stretched but in columns II and III it was. Columns I and II present measurements done with a screen attached to a shin, those in column III with a screen attached to the head.

TABLE I. SUBJECTS FROM 6 TO 14 YEARS OLD

SUBJECT	AGE (YEARS)	SEX	MASS (Kg.)	ν_s (C/S)		$\beta_s \cdot 10^{-4}$ (Gm. . Sec.$^{-1}$)	
				(F = 0)	(F = 25)	(F = 0)	(F = 25)
B	11	M	42	3.5	4.7	44	86
P	11	F	44	3.7	5.5	35	105
He	11	F	32	4.2	5.8	34	83
Ho	6	F	26	3.7	5.2	23	64
Ke	7	F	25	3.5	4.5	29	55
He	14	M	44	3.7	4.3	39	101
He	8	F	26	3.4	4.7	16	46
P	13	F	43	3.5	5.4	26	117
Ke	11	F	40	3.3	4.2	28	56
Kl	10	F	30	4.5	5.6	37	82
Ku	11	F	33	4.2	5.3	29	75
Mean	10		35	3.7	5.0	31	79

From the Tables I and II it appears that β_s and ν_s increase with increasing F. The increase on a stretched canvas is smaller than on a nonstretched one. Moreover, the differences in vibration properties of leg and head appear to be small, so they are strongly coupled.

Together with the measured values of β_s and D_s all constants necessary for the numerical calculations of amplitude and phase characteristics are known. We made calculations from these data for various circumstances for the following types of bcg's:

A. The *low-frequency*, critically ($\delta = 1$) and less than critically ($\delta = 0.4$) damped bcg with a (loaded or not loaded) natural frequency of 0.3 c/s (Figs. 3 to 16). (The ratio between the applied damping and the critical is called δ.)

TABLE II. SUBJECTS FROM 17 TO 54 YEARS OLD

SUBJECT	AGE (YEARS)	SEX	MASS (Kg.)	ν_s (C/S)						$\beta_s \cdot 10^{-4}$ (Gm. . Sec.$^{-1}$)					
				I (F = 0)	II (F = 0)	III (F = 0)	I (F = 25)	II (F = 25)	III (F = 25)	I (F = 0)	II (F = 0)	III (F = 0)	I (F = 25)	II (F = 25)	III (F = 25)
Me	24	M	65	3.2	—	—	4.5	—	—	40	—	—	180	—	—
Ho	18	F	55	3.3	4.1	4.5	4.6	5.6	5.1	25	46	98	130	64	65
Wa	20	F	80	3.3	3.9	4.6	4.8	—	4.4	71	62	70	170	67	160
Bu	22	M	74	3.2	—	5.5	4.4	4.2	4.3	90	70	105	170	86	85
He	24	M	76	2.9	—	—	4.0	—	—	53	—	—	150	—	—
V	22	M	76	3.0	—	—	4.3	—	—	43	—	—	200	—	—
Bo	18	M	85	2.9	4.2	4.9	4.3	5.1	—	55	78	95	185	100	—
T	18	M	66	3.2	4.4	4.0	4.1	5.1	4.4	65	78	83	115	110	50
J	20	F	51	3.0	—	—	4.0	—	—	48	—	—	85	—	—
Sc	17	M	65	3.4	4.4	4.0	4.6	5.8	5.7	48	75	45	62	83	95
Rd	22	M	93	—	3.9	3.8	—	4.2	5.4	—	67	54	—	120	72
A	21	M	77	—	3.8	4.3	—	3.8	5.0	—	58	45	—	87	90
We	21	M	60	—	3.7	3.6	—	5.1	4.1	—	65	50	—	130	110
Hm	22	M	70	—	4.1	4.0	—	6.1	5.7	—	73	64	—	78	51
Hk	26	M	81	3.2	4.0	4.0	4.2	4.1	3.9	64	69	69	145	120	100
Br	38	M	90	3.2	4.5	3.6	4.2	4.2	4.1	55	130	90	190	130	110
Be	54	M	68	3.5	4.7	4.0	4.9	4.9	4.5	27	70	48	205	110	90
St	41	M	74	3.4	—	4.0	4.4	4.4	4.1	39	—	54	100	94	65
Se	29	M	84	3.6	3.5	3.9	4.0	4.5	4.1	44	46	50	120	155	120
Ry	48	M	81	3.2	—	—	4.5	4.3	—	57	—	—	130	115	95
Bt	27	M	67	3.9	4.8	4.7	5.3	3.8	6.9	39	100	94	115	100	120
Ma	37	M	84	3.7	—	—	5.5	—	—	54	—	—	170	—	—
Mean			74	3.3	4.2	4.2	4.5	4.7	4.7	50	72	70	146	103	92

TABLE III. SUBJECTS FROM 17 TO 54 YEARS OLD. MEASUREMENTS ON A RIGID UNDERLAYER

SUBJECT	AGE (YEARS)	SEX	MASS (Kg.)	ν_s (C/S)				$\beta_s \cdot 10^{-4}$ (Gm. . Sec.$^{-1}$)			
				LEG (F = 0)	HEAD (F = 0)	LEG (F = 25)	HEAD (F = 25)	LEG (F = 0)	HEAD (F = 0)	LEG (F = 25)	HEAD (F = 25)
Ho	18	F	55	3.0	3.8	5.4	4.5	70	74	160	200
Wa	20	F	80	3.8	3.9	5.1	6.2	93	87	120	160
Bu	22	M	74	5.0	5.4	5.6	7.0	130	120	160	150
Bo	18	M	85	3.0	3.1	5.0	6.0	78	110	130	150
T	18	M	66	3.7	3.9	5.6	5.3	70	56	160	140
Sc	17	M	65	3.3	3.3	5.2	5.2	65	67	190	130
Rd	22	M	93	3.2	3.4	4.4	5.1	64	78	160	190
A	21	M	77	3.5	3.2	5.5	6.0	80	74	200	195
We	21	M	60	3.6	4.1	6.0	5.5	64	77	145	140
Hm	22	M	70	4.0	4.5	5.9	5.3	97	69	125	110
Hk	26	M	81	2.7	3.5	4.4	4.3	65	85	105	100
Br	38	M	90	3.4	3.2	5.7	4.1	95	120	150	165
Be	54	M	68	4.1	4.5	6.0	6.0	68	75	97	110
St	41	M	74	2.7	3.1	4.2	3.4	62	69	140	105
Se	29	M	84	3.4	3.2	5.8	5.1	82	90	210	155
Ry	48	M	81	3.0	3.1	4.2	4.3	73	—	—	—
Bt	27	M	67	3.5	3.3	5.0	5.5	77	72	215	185
Mean			75	3.4	3.7	5.2	5.2	78	82	154	149

TABLE IV

TYPE OF bcg	A. LOW-FREQUENCY							B. MIDDLE-FREQUENCY		C1. HIGH-FREQUENCY (STARR)		C2. HIGH FREQUENCY (DOCK)	
SUBJECTS	I	II	III	IV	V	VI	VII	VIII	IX	X	XI	XII	XIII
	(CHILDREN)			(ADULTS)		(ADULTS)		(ADULTS)		(ADULTS)		(ADULTS)	
	←Canvas not stretched - - - - - - - - - →←- - - - - - - - canvas stretched - - - - - - - - - - → rigid underlayer												
F (Kg.) (10^6 dynes)	0	0	25	0	25	0	25 .	0	25	0	25	0	25
m_b (Kg.)	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	15	15	—	—
m_s (Kg.)	35	35	35	74	74	74	74	74	74	74	74	74	74
D_b (10^5 Gm.. sec.$^{-2}$)	1.5	1.5	1.5	2.9	2.9	2.9	2.9	72	72	80.10^2	80.10^2	∞	∞
D_s (10^6 Gm.. sec.$^{-2}$)	19	19	35	33	60	52	65	52	65	52	65	37	80
β_b (10^3 Gm.. sec.$^{-1}$)	154	62	62	122	122	122	122	76.10^2	76.10^2	96	96	—	—
β_s (10^4 Gm.. sec.$^{-1}$)	31	31	79	50	146	71	98	71	98	71	98	80	150

B. The *middle-frequency*, more than critically damped ($\delta = 5$) bcg. The natural frequency, if there were no damping, has been chosen 1.5 c/s (Figs. 17 and 18).

C1. The *high-frequency*, far less than critically damped ($\delta = 6.10^{-3}$) bcg with a loaded natural frequency of 15 c/s, according to Starr (Figs. 19 and 20).

C2. The *high-frequency* bcg, according to Dock (Fig. 21).

All data used are tabulated in Table IV.* The value of β_b for the high-frequency bcg, according to Starr, was derived from measurements on the free vibration of a high-frequency bcg, loaded with dead weights, done by Bouhuys.[16]

DISCUSSION

As has been mentioned earlier in this paper we have assumed that an amplitude characteristic has to be flat with a deviation less than 10 per cent in the frequency range one is interested in (about 1 to 30 c/s). The corresponding phase characteristic is considered to be acceptable if the phase shift is less than 20 degrees.

A discussion of the characteristics presented yielded the following conclusions:

A. *The Low-Frequency bcg.*—(Figs. 3 to 16; in all figures the ordinates are represented in the C.G.S. system.)

1. The amplitude characteristic with critical damping of the loaded bcg does not meet the given requirement (Fig. 3). The damping will therefore further be chosen so small that there is 25 per cent overshooting ($\delta = 0.4$).

2. In all cases with $\delta = 0.4$, amplitude and phase characteristics concerning children and adults meet the requirements, provided the BCG is found by recording the displacement of the subject itself (Figs. 4 to 9). However, the characteristics improve by making use of a footplate that exerts a force ($F = 25$ Kg.) on the feet. The stretching of the canvas appeared to be of no importance in these cases.

3. When the BCG is found by recording the displacement of the bcg (Figs. 10 to 13) the characteristics do not fully meet the given requirements. Then the use of a footplate with $F = 25$ Kg. appears to be very important. The canvas being stretched or not appeared not to be important. If the footplate is used it is only for high frequencies (Fig. 13) that the characteristics do not meet the requirements.

4. Fig. 14 gives the characteristics of the bcg applying to the case when the bcg is not damped at all ($\beta_b = \delta = 0$). It turns out that the representation of the highest frequencies does not become better, whereas oscillations in the natural frequency (0.3 c/s) are very inconvenient. So, there is no reason to damp the low-frequency bcg to a lesser extent than corresponding to $\delta = 0.4$.

5. If the mass of the bcg m_b is made zero, there is still a small distortion of the higher frequencies (Fig. 15). The characteristics then meet the given requirements. So, if we wish to find the BCG by recording the displacement of

*In a preceding paper[4] the mass of the Starr bcg was erroneously stated to be 70 Kg.

Fig. 3.

Fig. 3.

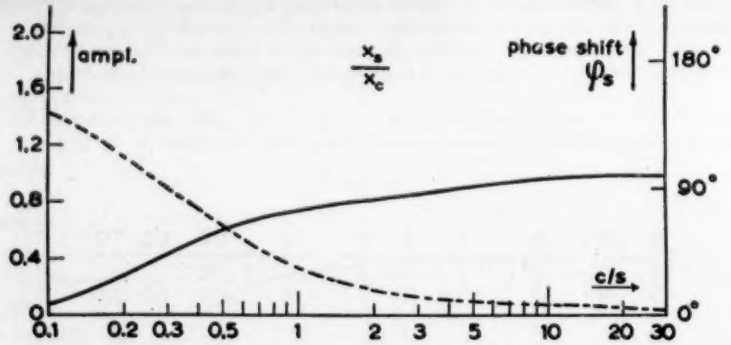


Fig. 4.

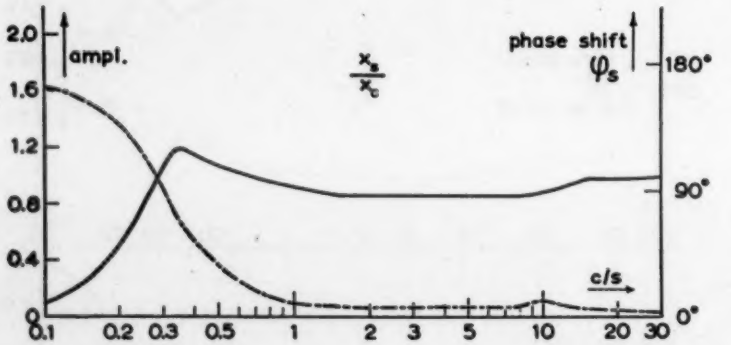


Fig. 5.

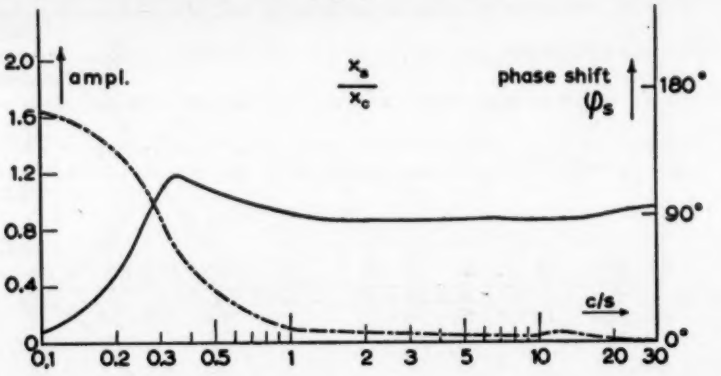


Fig. 6.

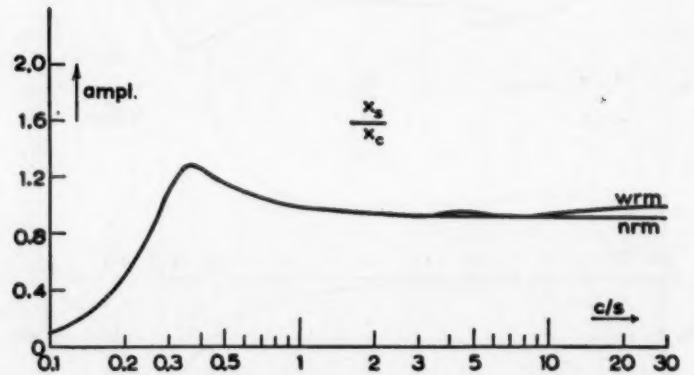


Fig. 3.—Amplitude and phase characteristics of the low-frequency, critically damped ($\delta = 1$) bcg, if the BCG is found by recording the displacement of the body of a child. No footplate. Canvas not stretched.

Fig. 4.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of a child. No footplate. Canvas not stretched.

Fig. 5.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of a child. With footplate. Canvas not stretched.

Fig. 6.—Amplitude characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of an adult. No footplate. Canvas not stretched. *wrm* = with relative movement, *nrm* = if there were no relative movement.

Fig. 7.

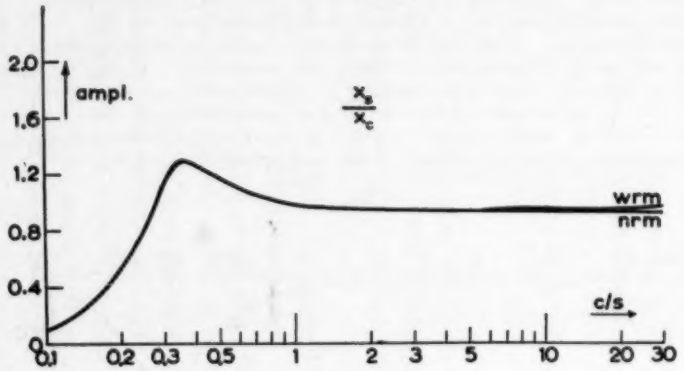


Fig. 8.

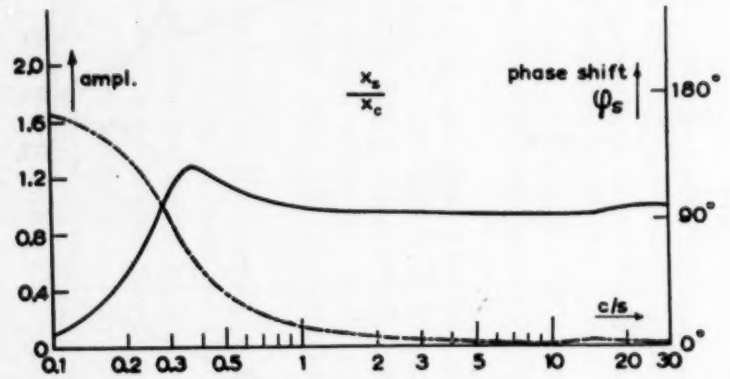


Fig. 9.

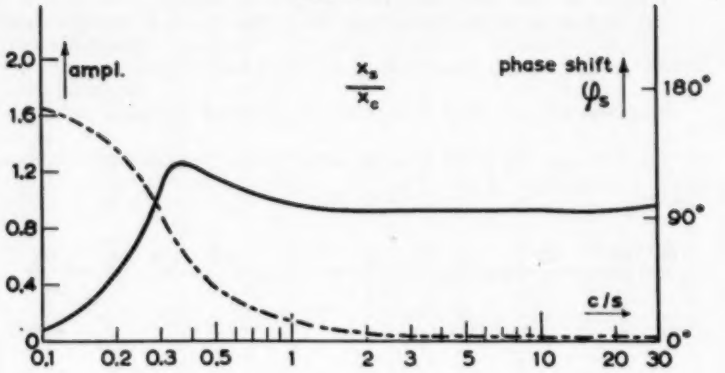


Fig. 10.

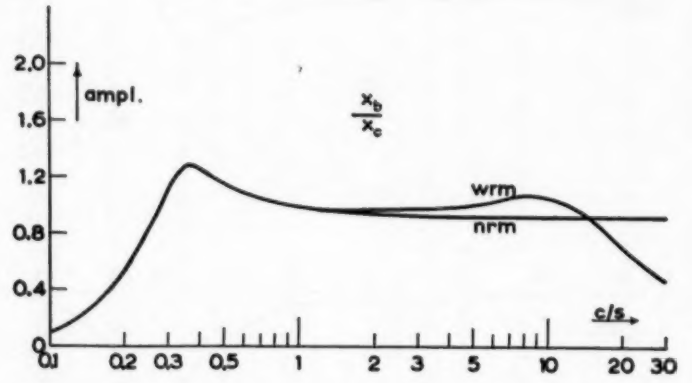


Fig. 7.—Amplitude characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of an adult. With footplate. Canvas not stretched. *wrm* = with relative movement, *nrm* = if there were no relative movement.

Fig. 8.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of an adult. No footplate. Canvas stretched.

Fig. 9.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the body of an adult. With footplate. Canvas stretched.

Fig. 10.—Amplitude characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the bcg. No footplate. Canvas not stretched. *wrm* = with relative movement, *nrm* = if there were no relative movement.

Fig. 11.

Fig. 13.

placement of the bcg. No footplate. Canvas not stretched. wrm =

Fig. 11.

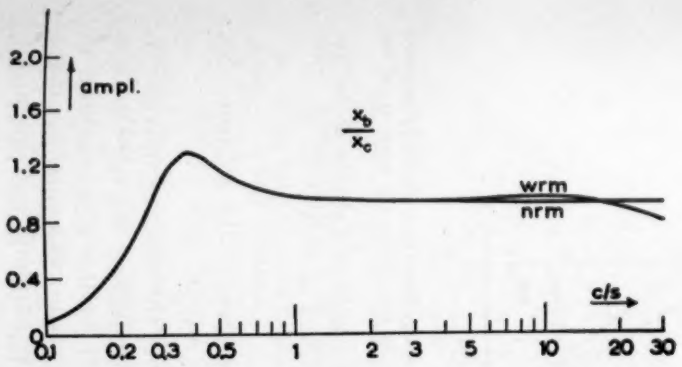


Fig. 12.

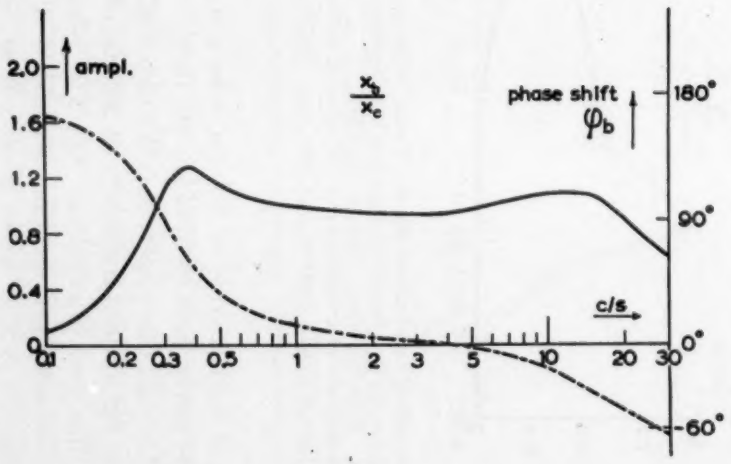


Fig. 13.

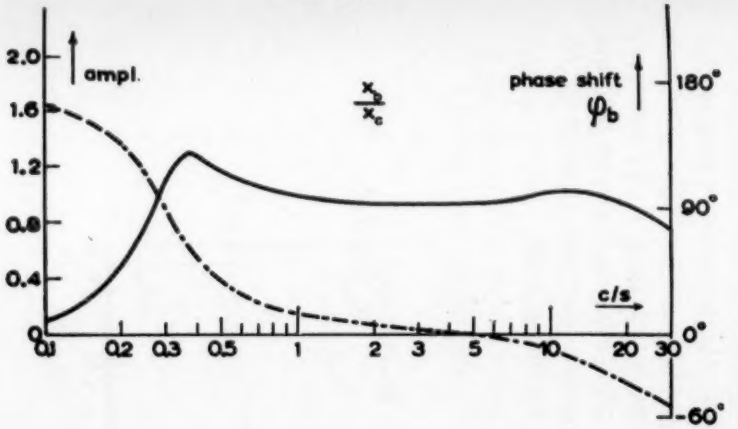


Fig. 14.

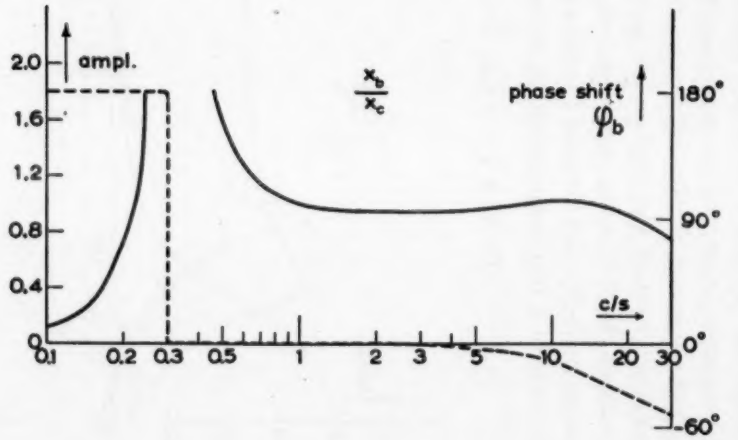


Fig. 11.—Amplitude characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the bcg. With footplate. Canvas not stretched. *wrm* = with relative movement, *nrm* = if there were no relative movement.

Fig. 12.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the bcg. No footplate. Canvas stretched.

Fig. 13.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the bcg. With footplate. Canvas stretched.

Fig. 14.—Amplitude and phase characteristics of the low-frequency, nondamped ($\delta = 0$) bcg, if the BCG is found by recording the displacement of the bcg. With footplate. Canvas stretched.

the bcg, it is necessary to make the mass, the damping, and the directive force of the bcg small enough. Our bcg has a mass of 6 Kg. which is mainly responsible for the fact that there is not a fully correct representation of the higher frequencies. That the distortion of the higher frequencies is not zero, if the mass m_b is zero, is clear from the following consideration: the coupling between bcg and surroundings is principally determined by the directive force per centimeter D_b and can, in the case of a bcg suspending on wires, be found from

$$D_b = 4\,\pi^2\,\nu_b^2\,(m_s + m_b).$$

Fig. 15.

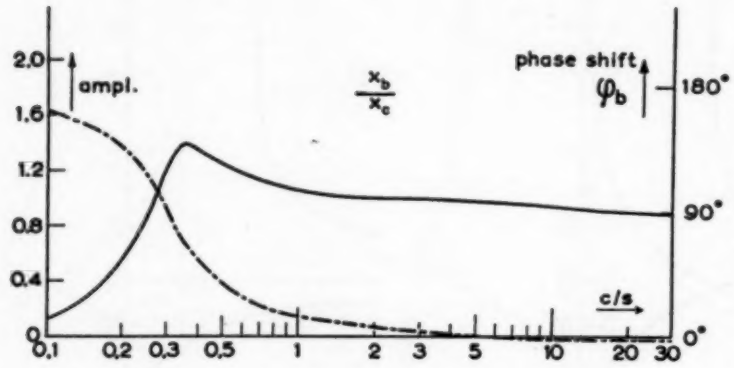


Fig. 16.

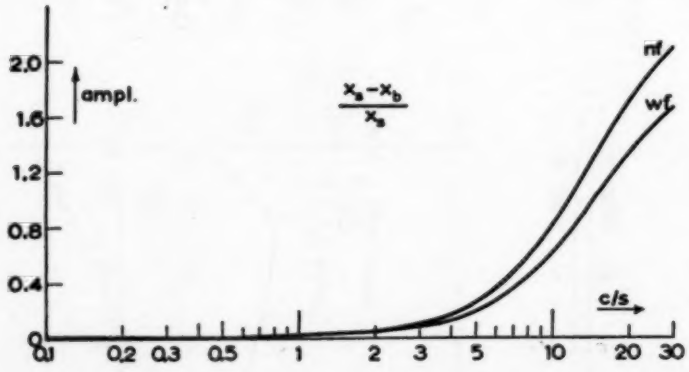


Fig. 15.—Amplitude and phase characteristics of the low-frequency, less than critically damped ($\delta = 0.4$) bcg, if the BCG is found by recording the displacement of the bcg, while the mass m_b of the bcg equals zero. With footplate. Canvas stretched.

Fig. 16.—Amplitude characteristics of the relative movement occurring with the use of the low-frequency, less than critically damped ($\delta = 0.4$) bcg. Canvas stretched. *nf* = no footplate, *wf* = with footplate.

For our bcg it holds that $\nu_b = 0.3$ c/s, where m_s is the mass of the subject. So, D_b cannot be made zero by making m_b zero.

6. In his valuable paper, von Wittern,[14] discussing the relative movement, has chosen as the natural frequency of his loaded low-frequency bcg $\nu_b = 0.6$ c/s. This choice has two drawbacks: (a) The phase shift in the neighborhood of 1 c/s is considerably greater than with $\nu_b = 0.3$ c/s; (b) The directive force per centimeter D_b is four times greater if $m_s + m_b$ has the same value and, therefore, the relative movement for the higher frequencies is greater. How

much greater can be calculated with the aid of formulas (4) and (5). Moreover, his bcg has a mass of 10 Kg., also making the relative movement greater than in our case. The use of shoulder clamps works in the opposite direction as point b and the large mass m_b of the bcg. Von Wittern found a natural frequency of the subject on the fixed bcg ν_s of 9 c/s. Working without shoulder clamps, however, is much less disturbing for the subject. In exchange for the omission of the shoulder clamps the natural frequency of the bcg ν_b must then be chosen at least about two times lower. Moreover, the phase shift in the neighborhood of 1 c/s is then acceptable.

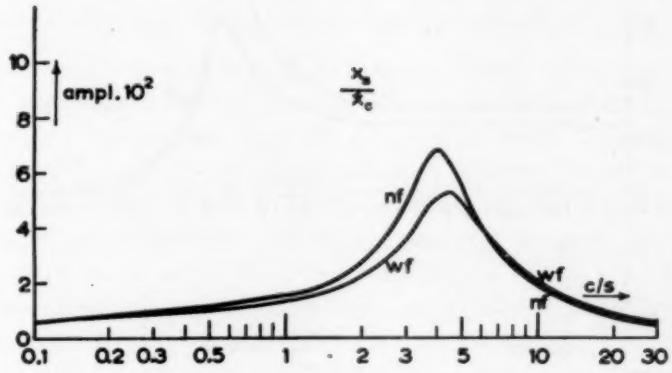


Fig. 17.

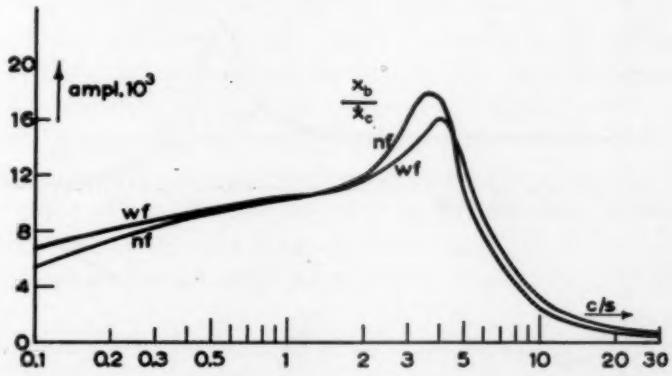


Fig. 18.

Fig. 17.—Amplitude characteristics of the middle-frequency, more than critically damped ($\delta = 5$) bcg, if the BCG is found by recording the displacement of the body of an adult. Canvas stretched. *nf* = no footplate, *wf* = with footplate.

Fig. 18.—Amplitude characteristics of the middle-frequency, more than critically damped ($\delta = 5$) bcg, if the BCG is found by recording the displacement of the bcg. Canvas stretched. *nf* = no footplate, *wf* = with footplate.

7. In Fig. 16 the amplitude of the difference in displacement of subject and bcg with respect to the amplitude of the subject is represented with the use of a footplate (curve *wf*) and without a footplate (curve *nf*). These curves cannot be found from the amplitude characteristics alone, for the phase difference between the movements must be taken into account. Formula (6) makes it possible to find the curves by substituting the known constants.

8. As can be seen, for instance, in Figs. 9 and 13 the flat parts of the amplitude characteristics are in the neighborhood of 3 c/s. The response, however,

is not 100 but about 93 per cent. This is caused by the fact that the mass m_b of the bcg is not zero, but 6 Kg. The amplitude in this range where the relative movement is negligible is therefore $m_s/m_s + m_b = 74/80$ times 100 per cent and equals 93 per cent. For proper calibration it is necessary to pay attention to this fact.

Fig. 19.

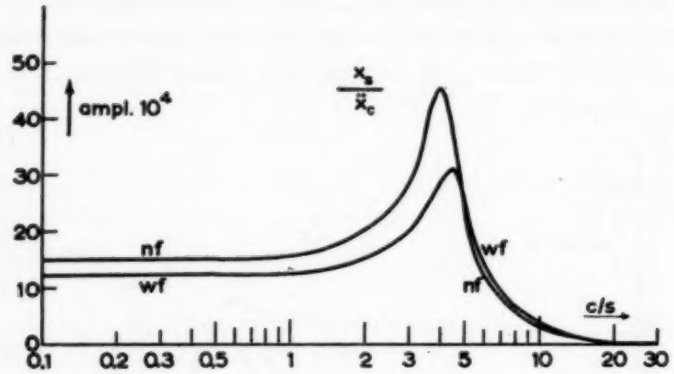


Fig. 20.

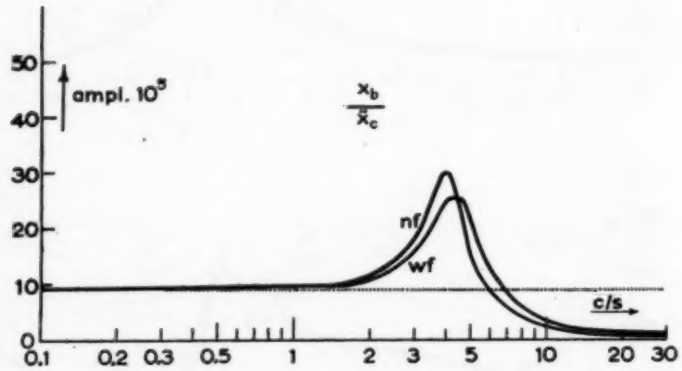


Fig. 21.

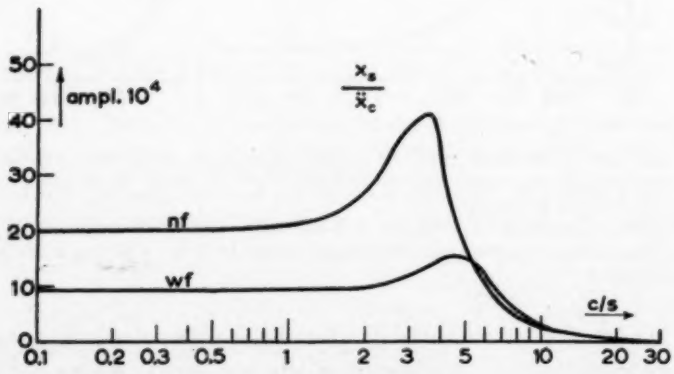


Fig. 19.—Amplitude characteristics of the high-frequency, far less than critically damped ($\delta = 6.10^{-3}$) bcg (Starr type), if the BCG is found by recording the displacement of the body of an adult. Canvas stretched. *nf* = no footplate, *wf* = with footplate.

Fig. 20.—Amplitude characteristics of the high-frequency, far less than critically damped ($\delta = 6.10^{-3}$) bcg (Starr type), if the BCG is found by recording the displacement of the bcg. Canvas stretched. *nf* = no footplate, *wf* = with footplate.

Fig. 21.—Amplitude characteristics of the high-frequency bcg (Dock type). *nf* = no footplate, *wf* = with footplate.

B. *The Middle-Frequency bcg.*—(Figs. 17 and 18.)

1. From the figures it appears that the amplitude characteristic whether a footplate is used or not, does not have a flat range in the frequency range one is interested in. In this calculation the damping is again chosen far more than critical ($\delta = 5$). The necessity of this appeared from an earlier investigation.[1] If the damping is chosen smaller, critical for instance, the amplitude distortion and the phase shift in the neighborhood of 1 c/s is not acceptable.

2. In point 1, it is shown how the displacement of the bcg and of the subject represents the velocity of the center of gravity $\dot{x}_c$.[1] Nickerson and Mathers[10] calculated the amplitude characteristic for the representation of the acceleration of the center of gravity $\ddot{x}_c$. They found a great distortion.[9,10]

C1. *The High-Frequency bcg According to Starr.*—(Figs. 19 and 20.)

1. From the figures it appears that the frequency characteristics are flat only for the lowest frequencies that are of interest and, thus, do not meet the assumed requirement. Both the characteristics holding good for recording the displacement of the subject and the displacement of the bcg have a maximum at about 4 c/s. The higher frequencies are scarcely represented. This can be seen from the BCG's recorded by a high-frequency bcg. The curves are very smooth; the so-called after vibrations have about the natural frequency of the subject with respect to the fixed bcg, i.e., about 4 to 5 c/s.

2. The sensitivity of this type of bcg is much smaller than that of the low-frequency bcg. This is caused by the strong binding of the bcg to the surroundings. If the acceleration $\ddot{x}_c$ is found by recording the displacement of the bcg, as is usual, the displacement of the bcg is less than 10 per cent of the displacement of the subject, caused by the large relative movement. For the lower frequencies (e.g., 1 c/s) this percentage equals the ratio D_a/D_b. In the latter case the sensitivity can be found by proper calibration, e.g., by exerting a constant external force of known amount on the subject and to measure the displacement of the bcg. The ratio D_a/D_b is then cancelled (dotted line in Fig. 20).

C2. *The High-Frequency bcg, According to Dock*[11].—(Direct-body method, Fig. 21.)

1. From the amplitude characteristics it appears that they greatly resemble the amplitude characteristics found for the Starr-type bcg (Figs. 19 and 20). So they, too, do not meet the requirement assumed for an amplitude characteristic.

2. When the Dock-type bcg is used, it is necessary to record the displacement of the subject, as the bcg is immovable. If the Starr-type bcg is used in the normal way, so that the displacement of the table is recorded, the Dock-type has a sensitivity of about 10 times that of the Starr-type.

3. The amplitude characteristic calculated by Smith and Rosenbaum[12] and by Smith, Rosenbaum, and Ostrich[13] for the Dock-type bcg, assuming that the leg-mounted transducer has no relative movement with respect to the legs, and our characteristics for this case (Fig. 21) agree fairly well. By making use of transducers with masses varying from 3 to 7 pounds, bound in several ways to the legs, these authors did not succeed in making the amplitude characteristic reliable.[12]

CONCLUSIONS

The amplitude and phase characteristics of the *low-frequency* bcg show that the deformation of a recorded BCG is negligibly small, especially when the displacement of the subject, instead of that of the bcg, is recorded. If a footplate is used the legs are fixed enough to record the movement of a shin. In this way the displacement of the center of gravity of the subject is recorded. The velocity and the acceleration of the center of gravity of the subject can be found by differentiating the displacement of the same *low-frequency* bcg once and twice, respectively. In the latter cases the amplitude and phase characteristics give a small deformation.[1] If the acceleration of the center of gravity is recorded in this way the respiration need not be held.

In contrast with the above the amplitude characteristics of the *middle-frequency* type and of the *high-frequency* types are not flat within the same limits.

Ballistocardiographic tracings with the same small deformation as occurring when making use of a *low-frequency* bcg, can possibly be found by the method proposed by Schwarzschild.[15] In that case, however, it is much simpler and easier to use a *low-frequency* bcg.

SUMMARY

Amplitude characteristics have been calculated numerically from formulas derived in this paper for the *low-frequency* bcg (Gordon, Henderson, Burger), the *middle-frequency* bcg (Nickerson), and the *high-frequency* bcg's (Starr and Dock), taking the difference in movement of subject and bcg into account. In a few cases corresponding phase characteristics have been calculated. Two constants of the system were determined experimentally.

From this it appears that it is only the *low-frequency* bcg that gives ballistocardiograms that are distorted slightly. Moreover, the velocity and the acceleration can be recorded with an analogously slight distortion.*

REFERENCES

1. Burger, H. C., and Noordergraaf, A.: Physical Basis of Ballistocardiography. II. The Quantities That Can Be Measured With Different Types of Ballistocardiographs and Their Mutual Relations, Am. Heart J. **51**:127, 1956.
2. Gordon, J. W.: On Certain Molar Movements of the Human Body Produced by the Circulation of the Blood, J. Anat. Physiol. **11**:533, 1877.
3. Henderson, Y.: The Mass-Movement of the Circulation as Shown by a Recoil Curve, Am. J. Physiol. **14**:287, 1905.
4. Burger, H. C., Noordergraaf, A., and Verhagen, A. M. W.: Physical Basis of the Low-Frequency Ballistocardiograph, Am. Heart J. **46**:71, 1953.
5. Nickerson, J. L., and Curtis, H. J.: The Design of the Ballistocardiograph, Am. J. Physiol. **142**:1, 1944.
6. Starr, I., Rawson, A. J., Schroeder, H. A., and Joseph, N. R.: Studies on the Estimations of Cardiac Output in Man, and of Abnormalities in Cardiac Function, From the Heart's Recoil and the Blood's Impacts; the Ballistocardiogram, Am. J. Physiol. **127**:1, 1939.
7. Elsbach, H.: Klinische onderzoekingen met de laagfrequente ballistocardiograaf volgens Burger, Thesis, Leiden, 1954.
8. Smith, J. E., and Bryan, S.: Simultaneous Calibrated Recording of Displacement, Velocity, and Acceleration in Ballistocardiography, Am. Heart J. **45**:715, 1953.

*After the preparation of this paper we received the papers of Talbot and Harrison, who follow the same line of thought independently (S. A. Talbot and W. K. Harrison: Dynamic Comparison of Current Ballistocardiographic Methods. Part I, Circulation 12:577, 1955; Part II, Circulation 12:845 1955: Part III, Circulation 12:1022, 1955).

9. Burger, H. C., and Noordergraaf, A.: Physical Basis of Ballistocardiography. III, AM. HEART J. **51**:179, 1956.
10. Nickerson, J. L., and Mathers, A. J. L.: A Study of the Physical Properties of the Ballistocardiograph, AM. HEART J. **47**:1, 1954.
11. Dock, W., and Taubman, F.: Some Techniques for Recording the Ballistocardiogram Directly From the Body, Am. J. Med. **7**:751, 1949.
12. Smith, J. E., and Rosenbaum, R.: Studies of the Effect of a Second Degree of Freedom in Ballistocardiography, AM. HEART J. **46**:799, 1953.
13. Smith, J. E., Rosenbaum, R., and Ostrich, R.: Studies With the Displacement, Velocity, and Acceleration Ballistocardiograph in Aortic Insufficiency, AM. HEART J. **48**:847, 1954.
14. von Wittern, W. W.: Ballistocardiography With Elimination of the Influence of the Vibration Properties of the Body, AM. HEART J. **46**:705, 1953.
15. Schwarzschild, M. M.: Ballistocardiography With Electronic Elimination of the Influence of Vibratory Properties of the Body, Proc. Soc. Exper. Biol. & Med. **87**:509, 1954.
16. Bouhuys, A.: High-Frequency Ballistocardiography, Proc. Roy. Dutch Acad. Sc., Series C **55**:126, 1952.

AURICULAR ESCAPE DURING VAGUS STIMULATION AFTER CRUSHING SINUS NODE TISSUE

D. SCHERF, M.D., S. BLUMENFELD, M.D., E. REID,* M.D., AND A. WEISZ, M.D.

NEW YORK, N. Y.

IN THE mammalian heart faradic stimulation of the right vagus nerve in the neck causes, as a rule, inhibition of impulse formation in the atrium and standstill of the heart, while stimulation of the left vagus leads to the appearance of A-V block with slowing of the atrial rate. Each vagus exerts its main action on its own side and only stimulation of the left vagus with strong currents results in the same effect as stimulation of the right vagus, namely, inhibition of impulse formation in the atrium.

In the course of an experiment during which the sinus node had been crushed with a surgical clamp, it was found accidentally that faradic stimulation of the right vagus inhibited only A-V conduction while the atria continued to beat regularly. Repetition of this experiment showed that this was not an isolated phenomenon and a systematic study of this form of "vagal escape" was undertaken.

METHOD

The experiments were performed on twenty-six mongrel dogs weighing between 8 and 15 kilograms. The animals were anesthetized with morphine (160 to 200 mg.) and Nembutal (60 to 180 mg.) administered intraperitoneally. After establishing artificial respiration with endotracheal intubation, the sternum and adjacent portions of the ribs were removed and the pericardium was opened. The vagus nerves in the neck were isolated but not severed and were stimulated with the aid of a Cambridge inductorium. A clamp was placed in the sinus node area along or across the taenia terminalis. No attempt was made to clamp off the sinus node completely; according to our experience this has not been possible with the application of only one clamp; contrary to many statements in the literature this is difficult even when three clamps are used. Coronary sinus rhythm and other forms of A-V rhythm appear readily even with a single clamp because of a variety of factors; soon, however, sinus rhythm reappears.

The electrocardiogram was always taken in Lead II.

From the Department of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals.

This investigation was supported, in part, by a research grant from the National Heart Institute, United States Public Health Service.

Received for publication Feb. 15, 1956.

*Trainee, National Heart Institute.

RESULTS

Sinus rhythm was present at the beginning of all experiments. Following the application of the clamp over the sinus node region the form of the P waves usually changed. Occasionally they remained positive but became higher or lower; often they were diphasic (± in Table I) or inverted. Since a pacemaker in the lower part of the sinus node may lead to inverted P waves in Leads II and III, particularly in the presence of intra-atrial block[6] due to the clamping, we cannot assume that negative P waves indicate the presence of an A-V rhythm. In one experiment A-V rhythm with simultaneous activation of auricles and ventricles appeared. Table I reveals that the P wave remained positive after the clamping in twelve of the twenty-six experiments; it became inverted in eight and showed a biphasic pattern in five; in one experiment A-V rhythm with simultaneous activation of auricle and ventricle existed.

In all experiments stimulation of the right vagus nerve in the neck before application of the clamp led to complete standstill of the heart caused by complete inhibition of the sinus node. Within the short time of the stimulation the deeper ventricular centers, which are not under the influence of the vagus, did not escape. After application of the clamp vagal stimulation did not inhibit the prevailing center at all in eight experiments. The atrial rhythm continued at the same rate and only A-V block appeared (Figs. 2 and 3). In fifteen experiments more or less marked slowing of the prevailing rhythm was observed with A-V block, while in four slow sinus rhythm or A-V rhythm without A-V block appeared. As shown in Fig. 5,*B* in two experiments the atrial rhythm developed slowly during vagus stimulation (rhythm of development).

Fig. 1,*B* shows the sinus rhythm continuing during the stimulation of the vagus at a slightly slower rate than before the stimulation, the P waves showing only the change of form characteristic of a strong vagus effect, as described for the heart of the turtle[2] and dog.[10]

Fig. 2,*B* shows negative P waves and Fig. 3,*B* positive P waves, both persisting during vagal stimulation at rates only slightly slower than the original rates. As seen in Fig. 4,*B*, vagal stimulation in this case led to a continuous A-V rhythm with P waves occasionally visible after the QRS complexes. In four experiments the phenomenon demonstrated in Figs. 1 to 3 did not appear. In its place, vagal stimulation after the application of the clamp led to a slow sinus rhythm or, as in Fig. 4, to an A-V rhythm. Analysis of these cases showed that the animals involved were sick either with pneumonia, severe diarrhea, or some other kind of infection and the heart was in poor condition at the onset of the experiment.

It is evident from Figs. 1, 2, 3, and 5 that the form of the P waves changes during the stimulation of the vagus. They assume a diphasic form with a negative wave following the positive one. This is due to the accelerated repolarization during vagus stimulation (a deepening and shortening of the T_P wave) and is seen typically in the dog heart.[10] It persists for a few beats after the vagus stimulation ends (Figs. 2,*A* and 5,*A*) and is characteristically absent in the first P wave which appears after the end of a long vagal inhibition (Fig. 2,*A*) for reasons discussed elsewhere.[8] These changes indicate that a strong vagus

TABLE I. CHIEF TABULATED RESULTS OF ALL EXPERIMENTS

DATES	FORMS OF P WAVE AFTER CLAMPING SINUS NODE	EFFECT OF VAGAL STIMULATION AFTER CLAMPING SINUS NODE	RATE BEFORE VAGAL STIMULATION	RATE DURING VAGAL STIMULATION	CENTER ACTIVE DURING VAGAL STIMULATION	REMARKS
2- 8-55	±	Atrial rhythm persists, A-V block	158	125	Same	
2-15-55	−	Complete inhibition				Heart in poor condition, dilated
3- 1-55	±	Atrial rhythm persists, A-V block	250	214	Same	
3- 8-55	+	Atrial rhythm persists, A-V block	270 214 250	186 200 189	Other	
3-22-55	±	Atrial rhythm persists, A-V block	166 138	143 138	Same	
3-29-55	+	Atrial rhythm persists, A-V block	150 102 143	136 102 150	Same Same	
4- 5-55	+	Sinus bradycardia				Heart in poor condition, pneumonia
4-12-55	+	Atrial rhythm persists, A-V block	143	94	Same	
5- 3-55	+	Atrial rhythm persists, A-V block	250	150	Same	
5-10-55	−	Atrial rhythm persists, A-V block	166 189	166 189	Same	
5-17-55	−	Atrial rhythm persists, A-V block	142	100	Same	
5-24-55	+	Atrial rhythm persists, A-V block	213	200	Same	

5–31–55	+	Atrial rhythm persists, A-V block	176	86	Other	
6–14–55	−	Atrial rhythm persists, A-V block	200	176	Same	
6–21–55	+	Atrial rhythm persists, A-V block	214	214	Same	
6–28–55	−	Complete inhibition of auricles with ventricular escape				Heart in poor condition, pneumonia
10– 4–55	−	Atrial rhythm persists, A-V block	166	125	Same	
10–18–55	+	Atrial rhythm persists, A-V block	166	100	Same	
10–25–55	−	Atrial rhythm persists, A-V block	150 166	139 157	Same	
11– 1–55	−	Atrial rhythm persists, A-V block	158	158	Same	
11–15–55	+	Slow A-V rhythm				Pneumonia, abscess
11–22–55	±	Atrial rhythm persists, A-V block	139	55	Same	
11–29–55	+	Atrial rhythm persists, A-V block	120 139	120 139	Same	
12– 6–55	±	Atrial rhythm persists, A-V block	200	200	Same	
1–10–56		Atrial rhythm persists, A-V block	125	125	Same	
1–17–56	+	Atrial rhythm persists, A-V block	200	200	Same	

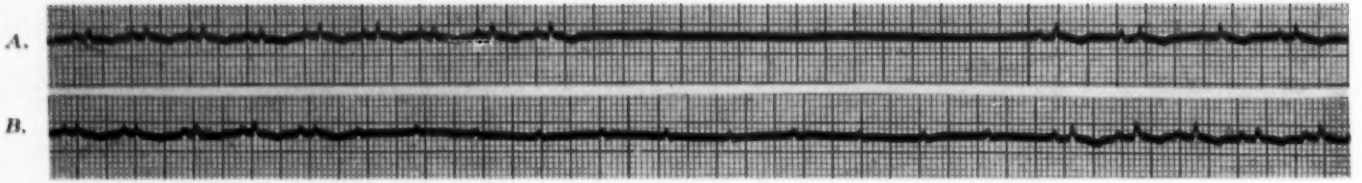


Fig. 1.—Experiment of March 29, 1955. *A* shows the effect of faradic stimulation of the right vagus before crushing sinus node tissue; *B*, after the crushing. The atrial rhythm persists, although slightly slowed during vagus stimulation in *B*. The P waves are not altered after the crushing.

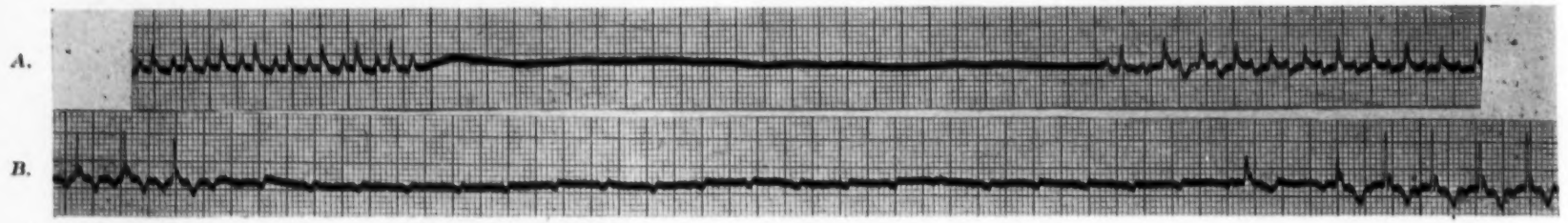


Fig. 2.—Experiment of May 10, 1955. *A* shows complete standstill of the heart during vagus stimulation. After the stimulation the P waves (with the exception of the first one) show the typical marked T_P waves lasting for 10 beats. After crushing, a rhythm with inverted P waves appears, and during vagus stimulation the ectopic rhythm persists at the same rate (166). During vagus stimulation the inverted P waves in *B* become smaller and show a positive T_P.

effect exists on the atria. When the P waves are negative at the start (Fig. 5,*B*) a positive T_P wave follows during vagus stimulation and this is easily confused with the injury current seen in atrial infarctions.

In most experiments, providing the animal was healthy, the auricular pacemaker remained the same before, during, and after the vagal stimulation.

This phenomenon of atrial escape from vagus inhibition was seen within a few seconds after the application of the clamp; in one case it occurred as early as eight seconds; and it persisted up to twenty-nine minutes after removal of the clamp.

In eleven experiments the clamp was applied first to different areas of the left or right atrium remote from specialized cardiac fibers. This procedure was without influence on the P waves and vagus stimulation caused only cardiac standstill. However, the atrial escape appeared as soon as the clamp was applied over the sinus node, and specific fibers crushed.

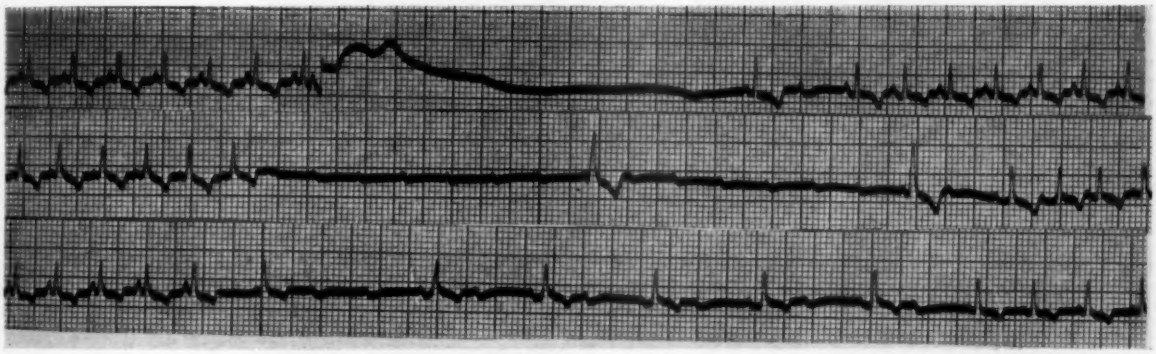


Fig. 3.—Experiment of Jan. 17, 1956. *A* shows complete standstill of the heart during vagus stimulation. *B*, taken after crushing, shows a change in the form of the P waves. During vagus stimulation the auricular rate remains unchanged (200). The P waves show the characteristic change of form. *C* reveals the effect of vagus stimulation twenty-two minutes after removal of the clamp. The auricular rate is slowed from 186 to 157.

DISCUSSION

Mechanical damage of specific tissue may lead to abnormal impulse formation in the damaged center. Firing of impulses has been observed in nerves and in the heart[9] following stretch or pressure and also following the appearance of injury currents due to more severe damage. They are, however, of short duration, lasting only a few minutes. Furthermore their rate is rapid and the picture caused by such mechanical damage is that of a paroxysmal tachycardia. Because in our experiments the cardiac rate before and after the application of the clamp was the same or only slightly faster and because the phenomenon discussed above lasted up to one-half hour after removal of the clamp, it is unlikely that a damaged center with increased excitability became the focus of impulse formation. We would rather assume that one of the uncrushed fibers of the sinus node became the pacemaker.

If we postulate the presence of a center with normal automaticity we must explain the lack of response or the slight response to vagal stimulation. In most experiments we crushed the upper part of the sinus node so that one may assume

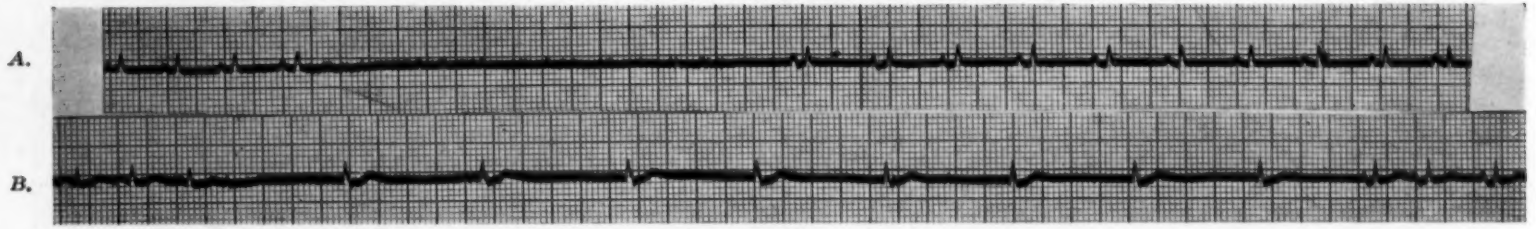


Fig. 4.—Experiment of Nov. 15, 1955. This animal had bilateral pneumonia and an abscess in the abdominal wall. The myocardium was very flabby and the heart was dilated. *A* shows cardiac standstill during vagus stimulation. In *B*, after crushing, stimulation of the vagus failed to produce auricular escape. A-V rhythm appeared.

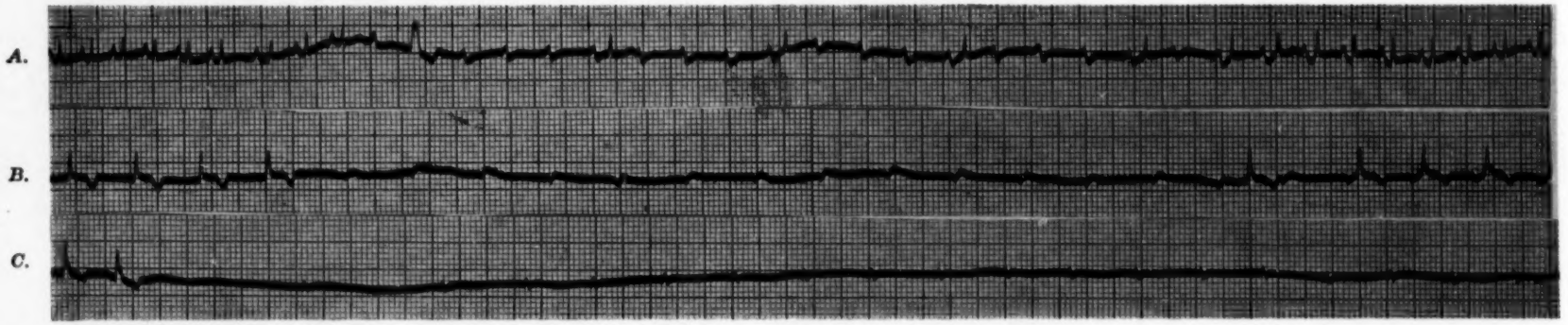


Fig. 5.—*A* is from the experiment on March 8, 1955. Vagus stimulation after application of the clamp shows continuation of the atrial rhythm with a rate falling from 270 to 186. The P waves appear biphasic because of a deeply inverted T_P. In *B*, from the experiment on Jan. 10, 1956, after application of the clamp the stimulation of the vagus did not change the rate of the atria (125 per minute). The inverted P wave is followed during vagus stimulation by a prolonged positive T_P wave, imitating the pattern occasionally seen in atrial infarction. In *C*, from the experiment of Feb. 8, 1955, the clamp had been removed fifteen minutes previously. Vagus stimulation does not inhibit the atria, but a slow rhythm of development is visible.

that the pacemaker was a center in the lower part of the sinus node. It may be of importance for the explanation of the phenomenon of vagal escape discussed in this paper that the concentration of sodium in the lower part of the sinus node is much less than in the upper part,[4] so that normally, with the pacemaker in the upper part, acetylcholine released during vagus stimulation causes complete inhibition, while it does not have this effect in the centers of the lower node. However, normally, without application of a clamp, the centers in the lower sinus node as well as the higher ones are inhibited by the vagus.

Crushing of muscle tissue causes release of potassium which changes the excitability of the heart. This factor must be discarded as an explanation since it would not account for the long persistence of the "vagal escape" after removal of the clamp.

In some experiments vagus stimulation slowed the atrial center while in others there was no effect; it is known that the effect of vagus stimulation in ectopic atrial rhythms, particularly auricular flutter, also varies. In flutter caused by electrical stimulation[3] and flutter elicited by topical application of aconitine,[8] faradic stimulation of the vagus may leave the rateun changed or may cause a marked increase.

There is also a clinical observation along the same line. In paroxysmal auricular tachycardia the various forms of vagal reflexes stop the tachycardia in about 50 per cent of the cases, while in the others no effect is noted. No explanation is yet known for this difference in response. It is possible that when the ectopic center is in the upper portion of the sinus node the response is better than when it is located in the lower portion of the node.

The experiments are also of interest in relation to results obtained by other authors after crushing of atrial tissue. It is well known that postfaradic flutter is elicited with difficulty in the auricle of a normal dog. Rosenblueth and Garcia Ramos[5] demonstrated that after crushing of a muscular bridge between vena cava superior, faradic stimulation of the atria elicits long lasting atrial flutter. This observation has been confirmed. According to these authors, crushing of the above-named muscular bridge converted vena cava superior and inferior into a single obstacle around which a circus wave can circulate for a long time. Lewis, however, found in most of his experiments a wave circulating around the venae cavae without crushing. We demonstrated that the same facilitation of post faradic flutter may be seen when a clamp is placed only over a part of this muscular bridge,[7] and Brown readily obtained flutter when "either an area of the auricular wall including some auricular and inferior vena caval tissue or a small area of the body of the right auricle was crushed."[1] Furthermore, were a circulating wave around the caval veins present, a broad ligature put across this path crushing all tissue in which a circus wave could possibly move, should interrupt the flutter. This is, however, not the case.[7] The flutter continues after the ligation without disturbance. It seems probable to us that with crushing specific fibers were injured which are brought more readily into a state of prolonged rapid firing of impulses by faradization. Studies of this question are in progress. It may well be that we are dealing here with another form of facilitation of ectopic rhythms by crushing of sinus node fibers.

Not enough facts are available to link the phenomenon described in this presentation with Wedensky facilitation, that is, the development of weak local depolarizations beneath a block in specific tissue created by crushing.

SUMMARY

The effect of faradic stimulation of the right vagus nerve was studied in twenty-six dogs after specialized fibers of the sinus nodes had been crushed by means of a clamp.

In eight experiments the atrial rate remained unchanged.

In fourteen experiments the atrial rate was slowed but not stopped.

In the remaining four experiments, the auricles were completely inhibited and the ventricles slowed. These animals were sick and the heart was in poor condition prior to the experiment.

The "vagal escape" of the atria appeared as early as eight seconds after the clamping and persisted after removal of the clamp for twenty-nine minutes. Clamping off of other parts of the atria not containing specialized fibers was without effect.

REFERENCES

1. Brown, B. B.: A Study of Factors Related to Effects of Quinidine in Experimental Auricular Flutter, Circulation **5**:864, 1952.
2. Churney, L., Ashman, R., and Biggins, C. H.: Effect of Vagus on the Monophasic Action Potential of Auricular Muscle, Proc. Soc. Exper. Biol. & Med. **70**:123, 1949.
3. Lewis, T., Drury, A. N., and Bulger, H. A.: Flutter and Fibrillation; The Effects of Vagal Stimulation, Heart **8**:141, 1921.
4. Oebrink, K. J., and Essex, H. E.: Chronotropic Effects of Vagal Stimulation and Acetylcholine on Certain Mammalian Hearts With Special Reference to the Mechanism of Vagal Escape, Am. J. Physiol. **174**:321, 1953.
5. Rosenblueth, A., and Garcia Ramos, J.: Studies on Flutter and Fibrillation. II. The Influence of Artificial Obstacles on Experimental Auricular Flutter, AM. HEART J. **33**:677, 1947.
6. Rothberger, C. J., and Scherf, D.: Zur Kenntnis der Erregungsausbreitung vom Sinusknoten auf den Vorhof, Ztschr. ges. exper. Med. **53**:792, 1954.
7. Scherf, D., and Blumenfeld, S.: Mechanism of Auricular Flutter Caused by Crushing and Electric Stimulation, Cardiologia **24**:193, 1954.
8. Scherf, D., Blumenfeld, S., Mueller, P., and Beinfield, W. H.: On the Response of Ectopic Auricular Tachycardias to Vagus Stimulation, AM. HEART J. **45**:95, 1953.
9. Scherf, D., Scharf, M. M., and Goklen, M. F.: Effect of Stretch and Pressure on Stimulus Formation in the Dog's Auricle, Proc. Soc. Exper. Biol. & Med. **70**:708, 1949.
10. Scherf, D., and Terranova, R.: Changes of the Ta Wave in Standard Leads Following Stimulation of the Vagus Nerves, Proc. Soc. Exper. Biol. & Med. **74**:353, 1950.

STUDIES OF PULMONARY HYPERTENSION

VII. Hemodynamic Effects of Acute Hypoxia in Patients With Mitral Stenosis

Paul N. Yu, M.D., David C. Beatty, M.D., M.R.C.P., Frank W. Lovejoy, Jr., M.D., Robert E. Nye, Jr., M.D., and Howard A. Joos, M.D.

Rochester, N. Y.

With the Technical Assistance of Ruth Knight and Carol Gouverneur

THE hemodynamic effects of hypoxia have been studied in normal subjects and in patients with various cardiopulmonary diseases by many investigators.[1-6] In most normal subjects during hypoxia the pulmonary artery pressure rises significantly, the cardiac output increases or does not change, the heart rate is more rapid, and the pulmonary "capillary" pressure is unaltered. Total pulmonary and pulmonary vascular resistances increase and the systemic artery pressure and peripheral vascular resistance decline.

The number of patients with mitral stenosis studied under similar conditions is still small.[3-5] It is the purpose of this report to describe the hemodynamic effects of breathing low oxygen concentrations in twenty-four such patients and to analyze the pulmonary pressure and the flow relationships during acute hypoxia and recovery.

CLINICAL MATERIAL AND METHOD

Twenty-four patients with predominant mitral stenosis were studied, none of whom had clinical or laboratory evidence of associated congenital cardiac lesions, chronic pulmonary disease, active rheumatic carditis, or other valvular lesions of significance. Thirteen patients were in sinus rhythm and eleven had atrial fibrillation during the study. All but three were studied before mitral valvuloplasty.

Cardiac catheterization was performed two or three hours after a light breakfast. Premedication with 0.1 Gm. sodium pentabarbital or 0.1 Gm. of Noludar* (Methyprylon [3, 3-diethyl-5-methyl-2, 4-piperidinedione]) was given in about half the cases. The catheter was advanced first into a distal

From the Chest Laboratory of the Department of Medicine of the University of Rochester School of Medicine and Dentistry and the Medical Clinics of Strong Memorial and Rochester Municipal Hospitals, Rochester, N. Y.

This study was supported in part by a research grant-in-aid from the National Heart Institute of the National Institutes of Health, Public Health Service, and by the Hochstetter Fund and Ernest L. Woodward Fund.

Received for publication Dec. 27, 1955.

*Courtesy of Hoffman La Roche Company, Nutley, N. J.

radicle of a pulmonary artery to occlude its lumen. After the pulmonary "capillary" pressure[7] was recorded the catheter was withdrawn until its tip was just distal to the bifurcation of the pulmonary artery. An indwelling needle (Gauge No. 19 Cournand or No. 20 Riley) was placed in a brachial or femoral artery. Oxygen consumption was measured by an open circuit method similar to that described previously.[8] When the ventilation and oxygen consumption were stable the cardiac output was determined by the direct Fick principle. Inspired gas was changed from room air to 12 per cent oxygen in nitrogen, delivered through an anesthesia bag and a three-way respiratory stopcock. The duration of hypoxia varied from ten to eighteen minutes, usually fifteen minutes or longer. Cardiac output was measured in the tenth or fifteenth minute of hypoxia and again, in some cases, after ten or more minutes of recovery breathing room air.

Pulmonary artery and peripheral artery pressures were recorded immediately after each determination of cardiac output, and pulmonary artery pressure was recorded every two minutes during hypoxia and recovery. Pulmonary "capillary" pressure was measured toward the end of the hypoxic period in thirteen cases by advancing the catheter and wedging it, after which the catheter was withdrawn again to the proximal pulmonary artery. In five cases a double lumen catheter was used, permitting simultaneous recording of pulmonary "capillary" and pulmonary artery, or of pulmonary artery and right ventricular pressures. In one of these the catheter was wedged throughout the study and after a twenty-minute interval, the study was repeated with the catheter ostia in pulmonary artery and right ventricle.

Pressures were recorded by means of a Statham pressure transducer connected to a carrier wave amplifier in a multichannel direct-writing oscillograph (Sanborn Poly-Viso Cardiette). The electrocardiogram and usually a pneumogram were recorded simultaneously. Pressure records were calibrated with a mercury manometer. Systolic and diastolic pressures were measured during at least two respiratory cycles and average values were calculated. Mean pressures were measured by planimetric integration of the tracings during one or two respiratory cycles. The arbitrary zero point of all pressures with the patient in a recumbent position was 6.5 cm. below the angle of Louis.

Blood oxygen content, capacity, and saturation were determined by the method of Van Slyke and Neill.[9] Arterial blood P_{O_2} was directly measured by the method of Riley and associates[10] and the mixed venous blood P_{O_2} was indirectly obtained from the oxygen dissociation curve. Peripheral and pulmonary resistances were calculated by the formulas described by Gorlin and associates.[11] The term pulmonary vascular resistance is substituted for pulmonary arteriolar resistance. Mitral valve flow and area were calculated by the formula of Gorlin and Gorlin.[12] Respiratory exchange ratio (R.Q.) was determined by duplicate analysis of samples of mixed expired or end-tidal air taken during the minute when the blood samples were withdrawn, using the Scholander 0.5 c.c. gas analyzer.

In a series of twenty cases studied under comparable conditions in our laboratory, duplicate determinations within twenty minutes showed R.Q. values of less than 1.0 in all but one patient, and a change in R.Q. from the first to the

TABLE I. STATISTICAL ANALYSIS OF TWO SUCCESSIVE DETERMINATIONS DURING CARDIAC CATHETERIZATION IN 36 PATIENTS STUDIED FOR DIAGNOSTIC PURPOSES

	FIRST DETERMINATION			SECOND DETERMINATION			RANGE OF DIFFERENCES BETWEEN TWO SUCCESSIVE MEASUREMENTS
	MEAN	S. E.	RANGE	MEAN	S. E.	RANGE	
Oxygen consumption (c.c./M.²/min.)	136	±4.1	90–174	135	±4.0	95–178	−10 to +13 (%)
A-V oxygen difference (c.c./L.)	56	±3	38–105	56	±3	37–104	−5 to +6 (c.c./L.)
Cardiac index (L./M.²/min.)	2.65	±0.13	1.09–5.04	2.66	±0.13	1.23–4.65	−9.6 to +12.6 (%)
Heart rate (beats/min.)	90	±4	52–155	89	±4	52–148	−12 to +8 (beats/M.min.)
Stroke index (c.c./M.²/beat)	35	±3.1	10–98	35	±3.2	10–98	−5 to +11 (c.c./M.²/beat)
Pulmonary artery mean pressure (mm. Hg)	34	±3.2	10–83	33	±3.5	9–84	−8 to +5 (mm. Hg)
Arterial blood oxygen saturation (%)	90.8	±0.1	70.6–98.9	90.7	±0.1	71.1–98.5	−3.4 to +2.8 (%)
Total pulmonary resistance (dynes-sec.-cm.$^{-5}$)	636	±75	170–1298	640	±73	152–1298	−28 to +19 (%)

second determination of less than 0.16 in eighteen cases. In a series of thirty-six patients studied for diagnostic purposes the variability of oxygen consumption, A-V oxygen difference, cardiac index, heart rate, stroke index, pulmonary artery pressure, arterial blood oxygen saturation, and total pulmonary resistance was determined in two successive measurements. The mean, standard error, and range of the two determinations and the range of difference between the second and the first are summarized in Table I.

The patients in this series were selected according to the following criteria (similar to those proposed by Fishman and his associates[4]):

A. The change in R.Q. from control to hypoxia or from hypoxia to recovery was less than 0.16.

B. The R.Q. during each of the entire three periods was less than 1.0.

C. The variation in oxygen consumption from control to hypoxia or from hypoxia to recovery was less than 13 per cent.

In order to compare the changes of each determinant from one period to the next, any deviation or variation exceeding the limits of variations listed in Table I is considered significant. For example, if the pulmonary artery pressure decreases more than 8 mm. Hg or increases more than 5 mm. Hg from control to hypoxia, or from hypoxia to recovery, the change is considered significant. The terms "increase," "decrease," and "no change" henceforth indicate that the change from one period to the next is or is not beyond these limits of variation in this laboratory. Successive determinations of the pulmonary "capillary" pressures were obtained in too few of the thirty-six control cases for statistical comparison.

RESULTS

The average pulmonary flow, pressures, and resistances during the three periods are shown in Fig. 1. The mean values, standard deviation, per cent change, and statistical significance of the changes from one period to the next are summarized in Table II.

A. *From Control to Hypoxia.*—

1. *Blood oxygen saturations and tensions:* Changes in the arterial and mixed venous blood oxygen saturation and tension were highly significant in most cases. The average arterial oxygen saturation was 91 per cent during control, declining to 70 per cent during hypoxia. Changes in the arterial blood oxygen tension were even more pronounced: 75 mm. Hg and 40 mm. Hg in the corresponding periods. The average mixed venous blood oxygen saturation fell from 64 per cent during control to 47 per cent during hypoxia. The reduction in the mixed venous blood oxygen tension was relatively small, inasmuch as the change occurred on the steep portion of the oxygen dissociation curve. The percentage and absolute changes in arterial or mixed venous blood oxygen saturation and tension did not correlate significantly with changes in pulmonary blood flow, pressures, or resistances.

2. *Blood flow:* The A-V oxygen difference was narrowed in thirteen cases, widened in three, and was unchanged in eight. The cardiac index was increased in ten cases, decreased in six, and remained unchanged in seven. Increase in the heart rate was noted in seventeen cases, decrease in only one case, with no change in the remaining six cases. The stroke index was increased in three cases, decreased in nine cases, and showed no change in eleven. In those cases in which the cardiac index increased, this was an effect of increased heart rate rather than stroke index. Statistical significance was shown for the average change in heart rate, but not for cardiac index or stroke index. There was no significant correlation between the absolute or per cent change in the mitral valve flow and the pulmonary artery pressure.

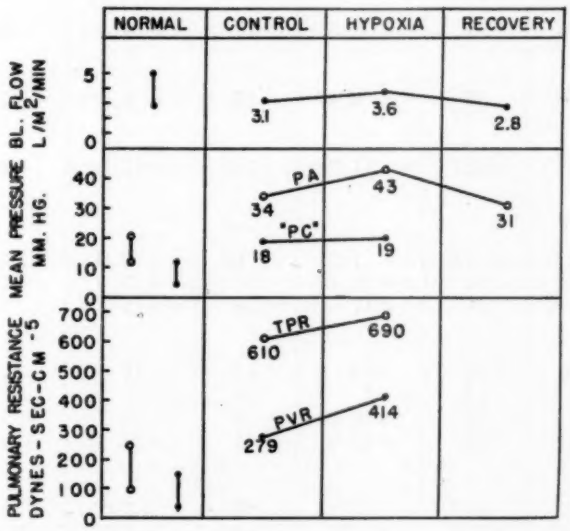


Fig. 1.—The average values of cardiac index, pulmonary artery and pulmonary "capillary" pressures, and total pulmonary and pulmonary vascular resistance in patients with mitral stenosis during the periods of control, hypoxia, and recovery.

3. *Blood pressures:* The pulmonary artery pressure rose in all twenty-four cases, significantly (more than 5 mm. Hg) in seventeen. The over-all change was statistically highly significant.

The rise in pulmonary artery pressure usually occurred within two minutes after beginning the low oxygen mixture, and continued for another five minutes, usually reaching a plateau before the eighth minute of hypoxia. As low oxygen was continued, no further rise in the pulmonary artery pressure usually occurred. The minute-to-minute change in the average of the pulmonary artery mean pressures is shown in Fig. 2. The pulmonary "capillary" pressure was measured in thirteen cases and its change was small and variable in direction.

The pulmonary artery-pulmonary "capillary" pressure gradient rose 6 mm. Hg. or more in nine out of thirteen cases. The gradient was increased less than 4 mm. Hg in two, remained unchanged in one, and decreased slightly in two. The average change was statistically significant.

TABLE II. STATISTICAL DATA IN

A. FROM CONTROL TO HYPOXIA

	NO. OF CASES	CONTROL		HYPOXIA		PER CENT CHANGE OF MEAN	p VALUE
		MEAN	S. D.	MEAN	S. D.		
Blood Gas Analysis							
$S_{a_{O_2}}$	24	91	± 3.45	70	± 7.1	−23	<0.01
$P_{a_{O_2}}$	24	75	±13.5	40	± 6.9	−47	<0.01
$S\bar{v}_{O_2}$	24	64	± 8.6	47	± 9.2	−26	<0.01
$P\bar{v}_{O_2}$	24	38	± 6.2	28	± 4.3	−26	<0.01
$C_{a_{O_2}} - C\bar{v}_{O_2}$	24	46.7	±11.7	41.9	±12.2	−10	0.01<p<0.05
Blood Flow							
C. I.	23	3.14	± 1.0	3.63	± 2.0	+16	>0.1
H. R.	23	81	±16.8	94	±19.6	+16	<0.01
S. I.	23	39	±17	39	±19	0	—
Pressures							
PA_m	24	34	±15	43	±17	+26	<0.01
PC_m	13	18	±7	19	±9	+5	—
PA_m–PC_m	13	16	±10.6	23	±15.7	+44	<0.01
BA_m or FA_m	18	95	±13.6	91	±15.5	−4	0.025<p<0.05
Resistances							
TPR	23	610	±426	690	±430	+13	0.025<p<0.05
PVR	13	279	±278	414	±330	+48	0.025<p<0.05
TSR	18	1540	±675	1370	±606	−11	—

$S_{a_{O_2}}$ = arterial blood oxygen saturation in per cent.

$S\bar{v}_{O_2}$ = mixed venous blood oxygen saturation in per cent.

$C_{a_{O_2}} - C\bar{v}_{O_2}$ = A-V oxygen difference in cubic centimeters per liter.

$P_{a_{O_2}}$ = arterial blood oxygen tension in mm. Hg.

$P_{\bar{v}_{O_2}}$ = mixed venous blood tension in mm. Hg.

In eighteen cases in which the systemic artery pressure was directly measured the mean pressure decreased 6 mm. Hg or more in nine cases. In four cases the decrease was less than 6 mm. Hg and in the remaining five there was an increase from 5 to 13 mm. Hg.

A positive correlation was observed between the per cent change of the pulmonary artery pressure and that of the total pulmonary resistance ($r = 0.63$, $p < 0.01$) and pulmonary vascular resistance ($r = 0.60$, $p < 0.05$). Similar correlation existed between the absolute change of the pulmonary artery pressure

HEMODYNAMIC EFFECTS OF ACUTE HYPOXIA

B. FROM HYPOXIA TO RECOVERY

	NO. OF CASES	HYPOXIA		RECOVERY		PER CENT CHANGE OF MEAN	p VALUE
		MEAN	S. D.	MEAN	S. D.		
Blood Gas Analysis							
$S_{a_{O_2}}$	16	72	± 7.6	91	± 5.2	+26	<0.01
$P_{a_{O_2}}$	16	39	± 7.3	73	±16.5	+87	<0.01
$S\bar{v}_{O_2}$	16	48	± 8.5	63	± 8.2	+31	<0.01
$P\bar{v}_{O_2}$	16	29	± 4.0	38	± 5.4	+31	<0.01
$C_{a_{O_2}} - C\bar{v}_{O_2}$	16	43.7	± 9.8	50.4	± 9.3	+16	<0.01
Blood Flow							
C. I.	15	3.21	±0.79	2.78	±0.50	−13	<0.01
H. R.	22	93	±18	82	±18	−11	<0.01
S. I.	15	35	±11	35	±9	0	—
Pressures							
PA_m	21	41	±17.5	31	±15	−24	<0.01
BA_m or FA_m	13	93	±14	97	±16	−4	—
Resistances							
TPR	14	700	±450	635	±464	−9	0.025<p<0.05
TSR	13	1385	±350	1589	±432	+15	—

H. R. = heart rate in beats per minute.
PA_m = pulmonary artery mean pressure in mm. Hg.
PA_m–PC_m = pulmonary artery to pulmonary "capillary" pressure gradient in mm. Hg.
TPR = total pulmonary resistance in dynes-sec.-cm.$^{-5}$.
PVR = pulmonary vascular resistance in dynes-sec.-cm.$^{-5}$.
TSR = total systemic resistance in dynes-sec.-cm.$^{-5}$.
C. I. = cardiac index in I./M.^{2}/min.
S. I. = stroke index in c.c./M.^{2}/beat.
PC_m = pulmonary "capillary" mean pressure in mm. Hg.
BA_m or FA_m = brachial artery or femoral artery mean pressure in mm. Hg.
The symbol "p" indicates the probability that this observed difference is a chance occurrence. Throughout this discussion differences with a chance probability of 0.05 or less are taken to be significant.

and that of the total pulmonary resistance ($r = 0.67$, $p < 0.01$) and pulmonary vascular resistance ($r = 0.76$, $p < 0.01$). There was no correlation between the percentage change in the pulmonary "capillary" pressure and any of the determinants. The absolute change in pulmonary artery to pulmonary "capillary" pressure gradient correlated directly with that of the pulmonary vascular resistance ($r = 0.88$, $p < 0.01$) and the correlation of the percentage change between these two determinants was less significant ($r = 0.58$, $p < 0.05$).

4. *Resistances:* The total pulmonary resistance was measured from control to hypoxia in twenty-three cases. It increased more than 20 per cent

in twelve cases and decreased more than 30 per cent in only one case in which the cardiac index during hypoxia was unusually high. In the remaining ten cases the deviation was not significant. The pulmonary vascular resistance was determined in thirteen cases from control to hypoxia. In eleven cases the pulmonary vascular resistance was definitely increased during hypoxia; in one case the increment was 200 per cent (Fig. 3). However, the over-all changes of both total pulmonary and pulmonary vascular resistances were just statistically significant. The response of total systemic resistance was variable and in most cases it was either decreased or unchanged.

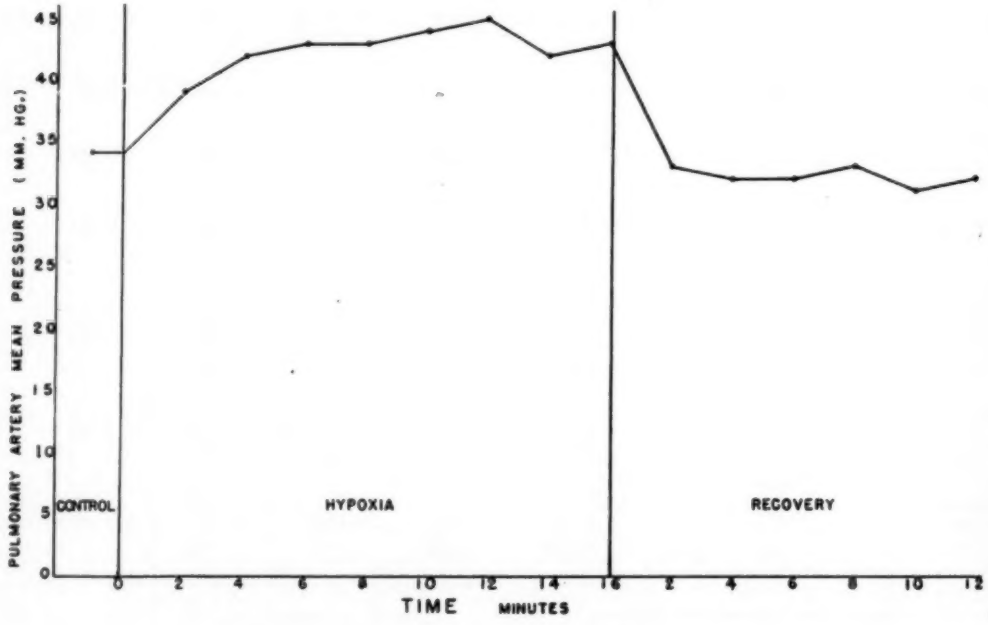


Fig. 2.—The minute-to-minute change in the average of the pulmonary artery mean pressures in twenty-four patients with mitral stenosis during the periods of control, hypoxia, and recovery.

B. *From Hypoxia to Recovery.*—

1. *Blood oxygen saturation and tension:* The arterial and mixed venous blood gas analysis was carried out in sixteen cases from hypoxia to recovery. About ten minutes after the cessation of 12 per cent oxygen breathing the blood oxygen saturation and tension returned to the control values. Again there was no significant correlation between percentage and absolute change in the blood oxygen saturation and tension and that of any of the following: cardiac index, stroke index, pulmonary artery pressure, and total pulmonary resistance.

2. *Blood flow:* In sixteen cases in which the A-V oxygen difference was determined during hypoxia and recovery it was increased in seven and unchanged in the remaining nine. In fifteen cases in which the cardiac index was determined there was a decrease in seven and no change in eight. The heart rate was decreased in fourteen cases and unchanged in eight. The stroke index was meas-

ured in fifteen cases. It was decreased in only three cases and was unchanged in twelve. The over-all changes in A-V oxygen difference, cardiac index, and heart rate were all statistically significant. There was no significant correlation between the per cent change in cardiac index and stroke index and changes in any of the following determinants: blood oxygen saturation, pulmonary artery pressure, pulmonary "capillary" pressure, pulmonary artery to pulmonary "capillary" pressure gradient, and total pulmonary resistance.

3. *Blood pressures:* There was an absolute decrease in the pulmonary artery pressure in all twenty-one cases in which it was measured. In ten cases the decrease was considered significant (more than 8 mm. Hg) and the average over-all change was highly significant. There was usually a precipitous decline of the pulmonary artery pressure within two minutes after the low oxygen mixture was discontinued. During the remaining period of recovery the pulmonary artery pressure remained fairly constant (Fig. 2). In many cases the pulmonary artery pressure during recovery was actually lower than that during control.

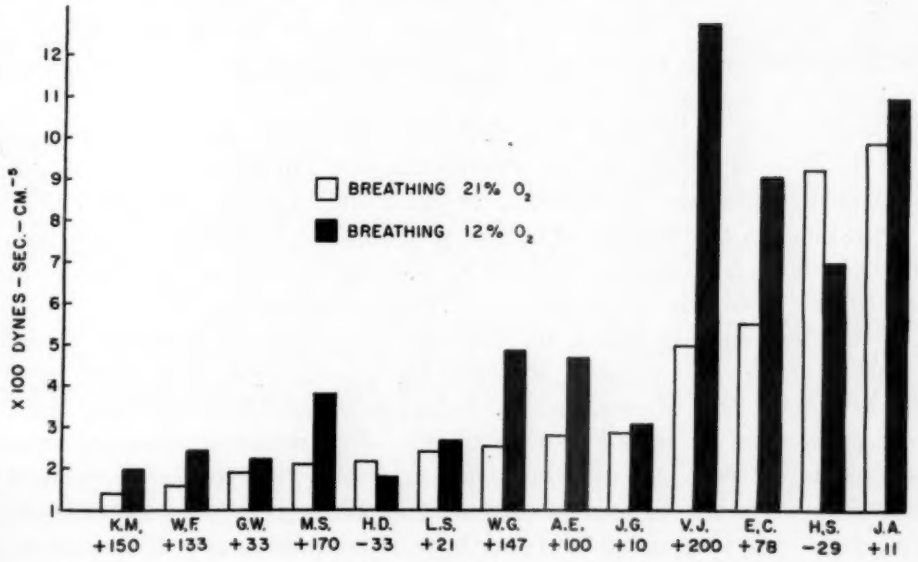


Fig. 3.—The pulmonary vascular resistance in thirteen patients with mitral stenosis during the periods of control and hypoxia. The numbers on the abscissa indicate percentage change from control to hypoxia.

Pulmonary "capillary" pressure was not measured during recovery, so the behavior of the pulmonary artery-pulmonary "capillary" pressure gradient from hypoxia to recovery was not demonstrated.

The change in the systemic artery pressure was variable. However, in most cases it rose slightly during recovery although an increase of 7 mm. Hg or more was noted in only six of thirteen cases.

4. *Resistances:* From hypoxia to recovery the total pulmonary resistance was reduced in ten of fifteen cases. In only two of these ten cases was the reduction 30 per cent or more. Of the remaining five cases three increased slightly and two remained practically unchanged. The trend of change in total systemic resistance was upward from hypoxia to recovery.

DISCUSSION

Acute hypoxia in patients with mitral stenosis induced a prompt, consistent, and usually significant elevation in mean pulmonary artery pressure in the present experiments. Fowler and associates[5] reported similar observations in mitral stenosis. Westcott and associates[2] and Doyle and associates[3] have found similar changes in normal subjects. On the other hand Fishman and co-workers[4] observed much less change in pulmonary artery pressure with acute hypoxia among patients with chronic pulmonary disease. The difference in response may reflect acclimatization of patients with chronic pulmonary disease to chronic hypoxia, insensitivity of chronically hypoxic oxygen receptors, or maximal response to a chronic low oxygen environment with inability to respond further to an added acute stimulus. Another explanation may be higher oxygen concentration administered to some of their cases than was given to ours.

The effect on pulmonary artery pressure in our patients was not apparently related to altered pulmonary "capillary" pressure. In contrast, a rise in both pulmonary artery and "capillary" pressures occurs almost invariably with exercise in patients with mitral stenosis, although pulmonary vascular resistance increases little or not at all.[13,14]

In most cases, pulmonary vascular resistance rose during acute hypoxia, reflected by a rise in pulmonary artery pressure and pulmonary artery to pulmonary "capillary" pressure gradient, with small and variable alteration in blood flow. However, in the absence of sufficient successive duplicate control determinations of pulmonary vascular resistance, the significance of its change during the experimental periods is speculative, although suggestive.

The mechanism responsible for the rise in pulmonary vascular resistance is obscure. Doyle and associates[3] favor the postulation that acute hypoxia results in constriction of precapillary pulmonary vessels as a direct effect of low oxygen tension in mixed venous blood. Experimental evidence suggests a vasoconstrictive response of pulmonary arterioles to hypoxia in animals.[15-18] Fishman and co-workers[19] recently administered 10 per cent or 12 per cent oxygen in nitrogen to one lung in patients with minimal pulmonary disease, but prevented marked anoxia of arterial and mixed venous blood by simultaneously administering 25 per cent or 30 per cent oxygen in nitrogen to the other lung. There was no rise in pulmonary artery pressure and no shift of blood flow from the anoxic to the hyperoxic lung, indicating that local anoxia does not produce pulmonary vasoconstriction. On the other hand, the consistent increase in pulmonary artery pressure and usually pulmonary vascular resistance in the present series suggests that acute hypoxia does result in functional pulmonary arteriolar vasoconstriction in patients with mitral stenosis, although the site of action is not clarified. Fishman and co-workers[19] have suggested that the increase in pulmonary artery pressure which attends acute bilateral pulmonary hypoxia may be passively produced by a shift of blood into the lung from the constricted peripheral vessels. In the presence of mitral stenosis, however, such a shift would be expected to increase the pulmonary "capillary" pressure also, without raising the pulmonary vascular resistance, which is contrary to our observations.

Recently, hexamethonium has been shown to lower the pulmonary artery pressure of many patients with mitral stenosis, possibly by pulmonary arteriolar dilatation secondary to a ganglionic blocking effect.[20,21] The combined effects of hypoxia and hexamethonium will be under study in the future. If hypoxia fails to augment pulmonary vascular resistance and pulmonary artery pressure in patients prepared with hexamethonium, we may suspect that the effect of hypoxia may at least in part be mediated through neurogenic vasomotion.

During the recovery period pulmonary artery mean pressure declined consistently. McGregor and associates[22] and Dressler and co-workers[23] have demonstrated a uniform decline in pulmonary artery mean pressure when patients with mitral stenosis breathed 100 per cent oxygen. The latter investigators point out that the fall in pulmonary artery mean pressure may result from reduction in the volume of blood flow through the pulmonary vascular bed, perhaps associated with a drop in left atrial and pulmonary venous pressure or from a decline in pulmonary vascular resistance. In the present study, cardiac index dropped in half of the cases during the recovery period. The over-all change was statistically significant. During the recovery period no measurements of the pulmonary "capillary" pressure were made, so that any change in the pulmonary vascular resistance could not be determined.

Of interest is the observation that acute hypoxia sufficient to reduce arterial and mixed venous blood oxygen saturation and tension consistently and significantly was not statistically related in magnitude to per cent changes in pulmonary flow, pressures, or resistances.

Hemodynamic responses to acute hypoxia have been studied in eleven additional patients with mitral stenosis. They are not included in the present report because of their more variable changes in R.Q. and oxygen consumption during the experimental periods. However, the effects of acute hypoxia on pulmonary blood flow, pressures, and resistances are similar to those reported.

SUMMARY AND CONCLUSION

1. Hemodynamic effects of acute hypoxia in twenty-four patients with mitral stenosis are presented.

2. During hypoxia more than two-thirds of the cases showed a significant increase in pulmonary artery pressure and heart rate. The pulmonary artery-pulmonary "capillary" pressure gradient rose 6 mm. or more in eight of thirteen cases. The arteriovenous oxygen difference was narrowed and the cardiac index increased in about half of the cases. The pulmonary vascular resistance rose in eleven of thirteen cases. The total pulmonary resistance tended to increase.

3. During recovery pulmonary artery pressure decreased more than 8 mm. Hg in ten of twenty-one cases. The arteriovenous oxygen difference was widened, and the cardiac index and heart rate decreased in about half of the cases. The total pulmonary resistance tended to fall.

4. The rise in the pulmonary vascular resistance, reflected by an increase in the pulmonary artery-pulmonary "capillary" pressure gradient out of proportion to blood flow suggests functional pulmonary arteriolar vasoconstriction, although the site of action is obscure.

We wish to express our appreciation and thanks to Dr. S. Lee Crump, Associate Professor of Radiation Biology and Scientist (Statistics), Atomic Energy Project, University of Rochester, for his help on the statistical aspects of this study, and to Mrs. Mary Sharp for preparing the manuscript.

REFERENCES

1. Motley, H. L., Cournand, A., Werko, L., Himmelstein, A., and Dresdale, D.: Am. J. Physiol. **150**:315, 1947.
2. Westcott, R. N., Fowler, N. O., Scott, R. C., Hauenstein, V. D., and McGuire, J.: J. Clin. Invest. **30**:957, 1951.
3. Doyle, J. T., Wilson, J. S., and Warren, J. V.: Circulation **5**:263, 1952.
4. Fishman, A. P., McClement, J., Himmelstein, A., and Cournand, A.: J. Clin. Invest. **31**:770, 1952.
5. Fowler, N. O., Cubberly, R., and Dorney, E.: Am. Heart J. **48**:1, 1954.
6. Yu, P. N., Lovejoy, F. W., Jr., Joos, H. A., Nye, R. E., Jr., Beatty, D. C., and Simpson, J. H.: Am. Heart J. **49**:31, 1955.
7. Hellems, H. K., Haynes, F. W., and Dexter, L.: J. Appl. Physiol. **2**:24, 1949.
8. Bruce, R. A., Lovejoy, F. W., Jr., Pearson, R., Yu, P. N. G., Brothers, G. B., and Velasquez, T.: J. Clin. Invest. **28**:1423, 1949.
9. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Vol. II, Baltimore, 1943, Williams & Wilkins Company.
10. Riley, R. L., Proemmel, D. D., and Franke, R. E.: J. Biol. Chem. **161**:621, 1945.
11. Gorlin, R., Haynes, F. W., Goodale, W. T., Sawyer, C. G., Dow, J. W., and Dexter, L.: Am. Heart J. **41**:30, 1951.
12. Gorlin, R., and Gorlin, S. G.: Am. Heart J. **41**:1, 1951.
13. Gorlin, R., Sawyer, C. G., Haynes, F. W., Goodale, W. T., and Dexter, L.: Am. Heart J. **41**:192, 1951.
14. Araujo, J., and Lukas, D. S.: J. Clin. Invest. **31**:1082, 1952.
15. Liljestrand, G.: Arch. Int. Med. **81**:162, 1948.
16. Nahas, G. G., Mather, C. C., Hemingway, A., and Visscher, M. B.: Am. J. Physiol. **167**:812, 1951.
17. Lewis, B. M., and Gorlin, R.: Am. J. Physiol. **170**:574, 1952.
18. Stroud, R. C., and Rahn, H.: Am. J. Physiol. **172**:211, 1953.
19. Fishman, A. P., Himmelstein, A., Fritts, H. W., Jr., and Cournand, A.: J. Clin. Invest. **34**:637, 1955.
20. Davies, L. G., Goodwin, J. F., and Van Leuven, B. D.: Brit. Heart J. **16**:440, 1954.
21. Balchum, O. J., Gensini, G., and Blount, S. G., Jr.: Clinical Research Proceedings **3**:115, 1955.
22. McGregor, M., Bothwell, T. H., Zion, M. M., and Bradlow, B. A.: Am. Heart J. **46**:187, 1953.
23. Dressler, S. H., Slonim, N. B., Balchum, O. J., Bronfin, G. F., and Ravin, A.: J. Clin. Invest. **31**:807, 1952.

CARDIAC METABOLISM IN EXPERIMENTAL VENTRICULAR FIBRILLATION

A. PEDERSEN, M.D.,* A. SIEGEL, M.S., AND R. J. BING, M.D.**

BIRMINGHAM, ALA.

RECENT studies on myocardial metabolism using catheterization of the coronary sinus have demonstrated that a reduction in coronary blood flow can result in disturbances in energy production.[1] It was shown, for example, that experimental hemorrhagic shock is accompanied by a significant diminution in myocardial extraction of pyruvate.[2,3] Thus, in most instances, the pyruvate level in coronary vein blood exceeded that of arterial blood. Despite increased arterial glucose concentration during oligemic shock, myocardial extraction and usage of glucose were reduced.[2] Most of these changes persisted after retransfusion of blood. It appeared possible that the trend toward negative myocardial pyruvate balance resulted from destruction of cocarboxylase (diphosphothiamine), a coenzyme which is indispensable for oxidative decarboxylation of pyruvate.[2] The present study was undertaken to investigate the effects of ventricular fibrillation resulting from electric shock.

MATERIALS AND METHODS

The studies were performed on a total of eight mongrel dogs. The animals were anesthetized with intravenous Pentothal sodium (30 mg. per kilogram weight). Immediately afterward, the animals were heparinized (400 mg. of heparin per kilogram weight). No additional heparin was administered during the experiment and nonheparinized syringes were used to sample the blood collected. These steps were taken since heparin, a sulfonated polysaccharide, interferes with the analyses of blood glucose, and since it was found that for several minutes following the intravenous injection of heparin the myocardial balance of glucose became negative. This may have been the result of incomplete mixing of heparin and blood, resulting in discrepancies in the glucose determination in individual blood samples. A tracheotomy tube was inserted and a femoral artery was cannulated for withdrawal of arterial blood samples. A No. 6 Birdseye catheter† was then introduced into an external jugular vein and inserted into the coronary sinus under fluoroscopic control.

From the Department of Experimental Medicine, The Medical College of Alabama, Birmingham, Ala.

Work supported by the U.S. Public Health Service, the Life Insurance Medical Research Fund, the American Heart Association, and the Wyeth Laboratories.

Received for publication Jan. 26, 1956.

*Fellow of the Egmont H. Petersen Foundation, Copenhagen, Denmark.

**Washington University Medical Service, VA Hospital, St. Louis, Mo.

†Available from U.S. Catheter & Instrument Corp., Glen Falls, N. Y.

TABLE I. METABOLIC CHANGES FOLLOWING ONSET OF VENTRICULAR FIBRILLATION (STATISTICAL ANALYSES)

DOG		OXYGEN VOL. % Δ	GLUCOSE		PYRUVATE		LACTATE		FATTY ACIDS		AMINO ACIDS		KETONES		POTASSIUM		SODIUM	
			ARTERIAL MG./100	Δ	ARTERIAL MG./100	Δ	ARTERIAL MG./100	Δ	ARTERIAL MG./100	Δ	ARTERIAL MG./100	Δ	ARTERIAL MG./100	Δ	ARTERIAL MEQ./100	Δ	ARTERIAL MEQ./100	Δ
1	B	11.3	90.0	3.5	.95	.250	13.00	5.61	—	—	—	—	—	—	4.3	.2	—	—
	A	5.0	88.3	1.3	.52	.050	17.76	.84	—	—	—	—	—	—	3.7	−1.2	—	—
2	B	9.7	88.0	1.6	.49	.080	12.95	5.72	.747	.012	4.60	.100	.017	.164	3.8	.6	147	0
	A	7.8	90.8	−13.4	.51	.005	21.88	−4.28	.757	.014	4.32	.040	.555	−.118	3.2	−2.1	143	0
3	B	13.0	88.1	5.8	.39	.040	13.35	6.25	—	—	—	—	.478	.097	—	—	—	—
	A	9.7	77.3	−12.2	.55	−.007	17.44	−.96	—	—	—	—	.511	0	—	—	—	—
4	B	11.6	88.7	1.8	.27	.070	23.50	8.95	.970	0	—	—	1.285	.200	3.5	.1	145	13
	A	12.2	80.0	−64.6	.59	.070	27.70	−11.60	.955	.010	—	—	1.170	−.115	4.0	−1.3	139	0
5	B	10.2	83.5	2.5	.40	.010	20.15	10.13	.870	.015	4.26	0	.835	.189	4.2	−.2	143	−6
	A	10.8	72.4	−3.2	.73	.070	24.70	−.43	.847	.002	4.44	0	1.133	.014	4.7	.2	151	4
6	B	11.8	80.7	6.6	.52	.080	17.35	8.77	1.240	.010	4.43	.180	.760	.120	4.0	−.1	149	−2
	A	11.5	74.9	−58.5	.50	−.050	24.00	−5.60	1.180	.030	4.59	0	.915	−.030	4.0	−4.7	149	−6

7	B	9.0	93.3	1.7	.45	.030	5.62	.69	.607	.002	2.92	0	1.700	.160	—	—	—	—
	A	4.4	90.5	1.0	.56	.030	10.26	1.61	.641	.025	3.03	.280	2.270	.450	—	—	—	—
8	B	10.8	90.4	6.3	.38	.040	10.80	2.12	—	—	4.00	.080	.756	.267	3.2	0	175	0
	A	5.5	92.8	3.2	.46	.006	17.88	.49	—	—	4.55	−.167	.681	−.260	3.2	−.58	175	−7
Mean of difference		−2.587	−4.463	−22.03	+.0713	−.0533	+5.61	−8.52	−.0108	+.0084	+.144	−.0414	+.2006	−.1794	−.0333	−1.713	−.40	−2.8
Range of observations		+4.2 to +12.2	+69.0 to +95.4	−149.3 to +4.6	+.36 to +.92	−.73 to +.76	+8.28 to +30.50	−28.9 to +2.66	+.607 to +.955	+.002 to +.030	+3.03 to +4.65	−.500 to +.280	+.245 to +2.760	−.570 to +.920	+3.15 to +4.70	−4.7 to +.2	139 to 175	−7 to +4
Degrees of freedom		7	7	7	7	7	7	7	4	4	4	4	6	6	5	5	4	4
t		2.66	2.28	2.25	.84	1.84	9.09	3.47	.20	1.29	1.09	.45	1.91	1.89	.17	2.40	.17	.73
p		$<.05$	$>.05$	$>.05$	$>.40$	$>.10$	$<.01$	$<.02$	$>.80$	$>.20$	$>.20$	$>.60$	$>.10$	$>.10$	$>.80$	$>.05$	$>.80$	$>.50$
Conclusions		S	NS	NS	NS	NS	S	S	NS	NS	NS	NS	NS	NS	NS	NS*	NS	NS

B = Before fibrillation (control).
A = After fibrillation.
Δ = Coronary arteriovenous oxygen difference.
S = Significant.
NS = Not significant.
*Almost significant.

TABLE II. METABOLIC EFFECTS FOLLOWING

DOG NO.	TIME (IN MINUTES)	RQ OF THE HEART	OXYGEN Δ (VOL. %)	ARTERIAL GLUCOSE (MG./100)	Δ GLUCOSE (MG./100)	ARTERIAL PYRUVATE (MG./100)	Δ PYRUVATE (MG./100)	ARTERIAL LACTATE (MG./100)	Δ LACTATE (MG./100)
	− 3	0.89	11.3	91.1	5.4	.85	.51	11.69	5.61
	− 2			89.0	1.6	1.05	0	14.31	.66
1	+ 2			89.0	−1.3	.46	.76	16.49	.66
June 3	+10	3.57	5.0	91.6	.2	.57	.07	17.31	−.50
	+15			84.1	2.7	.61	.09	18.20	.97
	+20			88.6	3.4	.45	−.73	19.05	2.22
	− 3	.90	9.7	88.3	1.7	.50	.05	13.30	5.78
	− 2			87.6	1.4	.48	.11	12.61	5.66
2	+ 2			94.8	2.5	.50	0	16.90	−2.08
June 4	+ 5	2.65	7.8	93.8	1.2	.45	0	18.60	−1.40
	+10			88.9	−8.0	.53	−.03	26.23	−4.58
	+12			85.8	−49.1	.54	.05	25.78	−9.04
	− 3	.79	13.0	88.1	7.1	.39	.03	13.29	5.95
	− 2			88.0	4.5	.38	.05	13.40	6.54
3	+ 5			82.7	2.1	.48	.02	13.33	−3.57
June 8	+13	1.31	9.7	80.3	−2.4	.43	−.28	15.39	−0.9
	+15			69.0	−36.2	.74	.24	23.6	1.6
	− 3	.95	11.6	88.6	1.6	.271	.07	23.8	8.8
	− 2			88.8	2.0	.271	.07	23.2	9.1
4	+ 5	.70	12.2	84.9	−.3	.52	0	24.2	−0.2
June 11	+ 8			82.4	−12.2	.44	−.08	26.5	−1.1
	+ 9			81.3	−96.7	.83	.51	29.4	−16.0
	+12			71.2	−149.3	.55	−.16	30.5	−28.9
	− 3	.88	10.2	86.9	3.9	.39	.02	19.1	+8.98
	− 2			80.0	1.0	.41	0	21.2	11.28
5	+ 4	1.38	10.8	72.9	−3.1	.54	.08	23.0	−2.1
June 21	+ 6			74.8	−1.2	.74	−.04	22.8	−.64
	+ 8			69.6	−5.2	.92	.18	28.3	1.46

ONSET OF VENTRICULAR FIBRILLATION

ARTERIAL FATTY ACIDS MEQ./100	Δ FATTY ACIDS MEQ./100	ARTERIAL AMINO ACIDS MG. N_2/100	Δ AMINO ACIDS MG. N_2/100	ARTERIAL KETONES MG./100	Δ KETONES MG./100	ARTERIAL K MEQ./100	Δ K MEQ./100	ARTERIAL NA MEQ./100	Δ NA MEQ./100
						4.3	.2		
						3.7	−1.2		
.747	.012	4.60	0.1	.017	.164	3.8	.6	147	0
.757	.014	4.32	.04	.245	−.390	3.2	−2.1	143	0
				.570	0				
				.850	.035				
				.460	.078				
				.497	.115				
				.575	−.075				
				.575	.075				
				.382	0				
.970	0			1.31	.28	3.5	+.1	145	13
				1.26	.12				
.955	.01			1.09	.05	4.0	−1.3	139	0
				1.14	.06				
				1.31	0				
				1.14	−.57				
.870	.015	4.26	0	.97	.324	4.2	+.2	143	−6
				.70	.054				
.847	.002	4.44	0	.97	.054	4.7	−.2	151	+4
				1.08	−.022				
				1.35	.011				

TABLE II.

DOG NO.	TIME (IN MINUTES)	RQ OF THE HEART	OXYGEN Δ (VOL. %)	ARTERIAL GLUCOSE (MG./100)	Δ GLUCOSE (MG./100)	ARTERIAL PYRUVATE (MG./100)	Δ PYRUVATE (MG./100)	ARTERIAL LACTATE (MG./100)	Δ LACTATE (MG./100)
6 June 24	− 3	1.04	11.8	81.0	6.2	.49	.05	17.4	8.05
	− 2			80.4	7.4	.56	.11	17.3	9.49
	+ 5	1.80	11.5	80.7	−3.0	.38	−.05	20.4	−1.8
	+10			69.0	−114.0	.62	−.05	27.6	−9.4
7 July 23	− 3	.79	9.0	94.9	3.3	.46	.02	6.15	.69
	− 2			91.6	0	.43	.04	5.08	−.25
	+ 7	1.53	4.4	88.6	1.2	.43	.02	8.28	.98
	+ 9			87.6	.6	.37	−.18	8.70	1.20
	+15			95.4	1.2	.87	+.24	13.81	2.66
8 Jan. 21, 1955	− 3		10.8	90.4	6.3	0.376	0.036	10.80	2.12
	− 2			90.4	6.3	0.376	0.036	10.80	2.12
	+ 1		4.2	92.2	1.5	0.358	0.072	17.05	1.60
	+ 3		5.6	94.6	3.6	0.508	0.035	17.05	1.64
	+ 5		6.7	91.6	4.6	0.508	−0.088	19.55	−1.77

With the catheters in place, coronary vein and femoral arterial blood were simultaneously collected for the determination of blood concentrations of oxygen, pyruvate, glucose, lactate, amino acids, fatty acids, and ketones.

Ventricular fibrillation was produced by electric shock with house currents (AC 120 volts) through two electrodes placed on a foreleg and a hind leg, respectively. One shock was usually sufficient to elicit fibrillation. Two sets of control data were collected, two or three minutes prior to the production of ventricular fibrillation. Since there was little or no free flow of blood through the coronary sinus, blood was sampled by pulling with a syringe attached to the catheter placed in the coronary sinus.

The extractions of oxygen and foodstuffs by the myocardium were calculated as the difference between their respective arterial and coronary sinus blood concentration. Blood glucose was determined by the method of Hagedorn and Jensen, using Somogyi's method[4] to prepare the blood filtrates. Pyruvate was analyzed with the method of Friedemann and Haugen[5] using a trichloracetic acid filtrate. Lactate was measured by the method of Barker and Summerson.[6] The manometric method of Van Slyke and Neill[7] was used for blood oxygen analyses. Fatty acids were determined according to the method of Man and Gildea, which represents a modification of the volumetric analysis of Stoddard and Drury.[8,9] Amino acids were analyzed according to the method of Albanese

—CONT'D

ARTERIAL FATTY ACIDS MEQ./100	Δ FATTY ACIDS MEQ./100	ARTERIAL AMINO ACIDS MG. N_2/100	Δ AMINO ACIDS MG. N_2/100	ARTERIAL KETONES MG./100	Δ KETONES MG./100	ARTERIAL K MEQ./100	Δ K MEQ./100	ARTERIAL NA MEQ./100	Δ NA MEQ./100
1.24	.01	4.43	.18	.82	.12	4.0	+.1	149	−2.0
				.70	.12				
1.18	.03	4.59	0	1.12	.47	4.0	−4.7	149	−6.0
				.71	−.53				
				1.78	.16				
.607	.002	2.92	0	1.62	0				
				1.62	.16				
.641	.025	3.03	.28	2.76	.92				
				2.43	.27				
		4.00	.08	0.756	0.267				
				0.756	0.267	3.15	0	175	0
		4.50	0	0.489	−0.356				
		4.65	0	0.847	−0.282	3.15	−0.55	175	−7.0
		4.5	−0.5	0.706	−0.141	3.15	−0.60	175	−7.0

and Irby[10] and ketones by a modification of the micromethod of Greenberg and Lester.[11] Serum potassium and sodium concentrations were obtained with the Beckman flame photometer. The method of Fiske and Subbarow was used to determine inorganic phosphorus.[12]

RESULTS AND DISCUSSION

Statistical analyses comparing averages of data collected during the control period and during the initial period of ventricular fibrillation illustrate that the significant changes in average values were a fall in myocardial oxygen extraction, an increase in the arterial lactate concentration, and a diminution in myocardial lactate extraction ($p < .02$) (Table I). The mean fall in lactate extraction by the heart muscle was 8.52 mg. per cent with a range of from −28.9 to +2.66 mg. per cent. Apparently ventricular fibrillation produced in heart muscle the classical response to anoxia, that of lactic acid production. The diminution in oxygen extraction by the heart muscle illustrates that active oxidation is seriously interfered with; no compensatory change in coronary flow is possible in these animals, since in ventricular fibrillation the cardiac output is reduced to at least 1/20 of its normal value.[13] With this diminution in cardiac output, coronary flow must be equally low.

Inspection of individual data shows changes affecting myocardial balances of all other metabolites and of sodium and potassium as well (Table II). Glucose extraction by the heart dropped an average of 22 mg. per cent, pyruvate of 0.0533 mg. per cent, amino acids of 0.0414 mg. per cent nitrogen, ketones of 0.1794 mg. per cent, potassium of 1.713 meq., and sodium of 2.8 meq. (Table I). Out of eight animals studied, six developed negative myocardial glucose and all showed negative myocardial pyruvate balance (Table II). Of six animals, all showed a higher potassium concentration in coronary vein than in arterial blood. This is probably the result of increased permeability or destruction of the cell membrane. Six of seven dogs developed a negative myocardial ketone balance and one of five showed negative amino acid balance (Table II). Sodium concentrations of coronary sinus blood were higher than those of arterial blood in two of five dogs with ventricular fibrillation (Table II). Only fatty acid uptake by the heart appeared to be unaffected (Table II). The magnitude of the changes observed was also considerable. For example, in four animals, glucose concentration in coronary vein blood during fibrillation exceeded that in arterial blood by more than 30 mg. per cent and in two animals by more than 100 mg. per cent (Table II).

The results demonstrate that ventricular fibrillation results in severe metabolic disturbances in energy production of heart muscle. It is likely that the increase in pyruvate concentration in coronary vein blood is the result of destruction of cocarboxylase. This coenzyme, diphosphothiamine, is indispensable for oxidative decarboxylation of pyruvate; under anaerobic conditions, the coenzyme is dephosphorylated.[14,15] The appearance of increased amounts of glucose is more difficult to interpret. The increase in lactate concentration in coronary vein blood is the result of glycolysis operating via the usual glycolytic scheme.

Increased content of inorganic phosphorus in coronary vein blood, which is frequently observed in these experiments, is likely to be the result of destruction of certain coenzymes, like adenosine triphosphate, which are maintained in their phosphorylated form only as long as active oxidation is assured.

It is likely that in the artificially perfused fibrillating heart or in the perfused heart in asystole, the metabolic changes are not as severe. Gregg has shown that if the coronary arteries are perfused, the induction of ventricular fibrillation invariably increases coronary inflow and coronary sinus outflow.[16] A similar situation pertains during asystole produced by vagal stimulation. It was further discovered that under these conditions the oxygen consumption of the myocardium was only moderately reduced as a result of diminished myocardial oxygen extraction. The increase in coronary flow and slight diminution in myocardial oxygen consumption during fibrillation and asystole persisted for thirty seconds following cessation of the normal beat. Gregg is of the opinion that myocardial oxygen consumption obtained under these conditions represents the resting metabolism of the heart.

Apparently, as long as adequate pressure head for the perfusion of coronary arteries is available, the myocardial oxygen consumption is only slightly reduced. In the experiments reported in this paper, however, the severe meta-

bolic disturbances encountered are likely to be the result of myocardial ischemia and hypoxia which are present under these experimental conditions as well as during ventricular fibrillation in men.

SUMMARY

The myocardial extractions of glucose, pyruvate, lactate, amino acids, fatty acids, ketones, sodium, and potassium were studied before and following experimental ventricular fibrillation in the dog.

Disturbances in myocardial balances of all of these substances were present. Particularly affected were the myocardial extractions of oxygen and lactate.

The significance of these findings was discussed.

REFERENCES

1. Bing, R. J.: Harvey Lecture Series, Series L, 1954-1955, Academic Press, p. 27.
2. Edwards, W. S., Siegel, A., and Bing, R. J.: J. Clin. Invest. **33**:1646, 1954.
3. Hackel, D. B., and Goodale, W. T.: Circulation **11**:628, 1955.
4. Hagedorn, H. C., and Jensen, B. N.: Biochem. Zeit. **135**:46, 1923.
5. Friedemann, T. E., and Haugen, G. E.: J. Biol. Chem. **147**:415, 1943.
6. Barker, S. B., and Summerson, W. H.: J. Biol. Chem. **138**:535, 1941.
7. Van Slyke, D. D., and Neill, J. M.: J. Biol. Chem. **61**:523, 1924.
8. Man, E. B., and Gildea, E. F.: J. Biol. Chem. **99**:32, 1932.
9. Stoddard, J. L., and Drury, P. E.: J. Biol. Chem. **84**:741, 1929.
10. Albanese, A. A., and Irby, V.: J. Lab. & Clin. Med. **30**:718, 1945.
11. Greenberg, L. A., and Lester, D.: J. Biol. Chem. **154**:177, 1944.
12. Fiske, C. H., and Subbarow, Y.: J. Biol. Chem. **66**:375, 1925.
13. Southworth, J. L., Pierce, E. C., II, Rawson, F. L., Jr., and McKusick, V. A.: Surgical Forum, Boston, 1950, p. 221.
14. Ochoa, S.: Biochem. J. **33**:1262, 1939.
15. Govier, W. M., and Greer, C. M.: J. Pharmacol. & Exper. Therap. **72**:321, 1941.
16. Gregg, D. E.: Shock and Circulatory Homeostatis. Transactions of the Fourth Josiah Macy, Jr., Foundation, December, 1954, Ed. H. D. Green.

A NEW LOOK AT ELECTROCARDIOGRAPHIC LEADS

ABRAHAM I. SCHAFFER, M.D., SERGE BLUMENFELD, M.D.,
ROBERT BUSSAN, M.D., AND DAVID SCHERF, M.D.

NEW YORK, N. Y.

OFFICIALLY the American Heart Association,[1] while recommending that V_1 through V_6 be registered routinely, does not give any specific advice as to what limb leads should be routine. This omission may indicate uncertainty as to what limb leads are needed. Recent advances in our understanding of the recording of the heart's electrical activity have prompted us to attempt to simplify the prevalent multiple lead techniques.[2] We have come to the conclusion that Leads I, aV_F, and the chest Leads V_1 through V_6 should be taken routinely. On the other hand, Leads II, III, aV_R, and aV_L are, for practical purposes, not needed. This is contrary to the present belief that a record is complete and the maximum information is supplied when, in addition to the chest leads, all six limb leads are recorded.

Present lead routines are the result more of a historical development than of modern theory and experience. The bipolar Leads I, II, and III were considered by Einthoven to record the heart's activity as if the heart were equivalent at any instant to a single, small dipole centered in a flat, homogeneous volume conductor. The electrodes on the limbs were assumed to lie at the apices of an equilateral triangle, and were treated as distant or indirect electrodes that recorded the activity of all parts of the heart equally well.

Unipolar leads were introduced by Wilson together with the assumption that the exploring electrode registered the activity of the adjacent myocardium more than of the distant regions. Thus, it was considered to function as a semidirect electrode with a local pick-up action. Applied to the precordial unipolar leads this interpretation proved very fruitful; so much so, that it is still a very popular method, although it has since been shown not to be generally valid except on infrequent occasions. Even the unipolar limb leads have been treated as semidirect leads, thereby resulting in many illogical conclusions.

As vectorcardiography became an important method it seemed for a time that it might even displace the electrocardiogram. Although it soon became clear that it could not compete clinically with the latter, it led to the vector interpretation of the electrocardiogram.[3-5] In this method both limb and precordial electrodes were treated as distant or indirect in their action. While it proved to be generally successful, at the same time it made it possible to recognize occasional instances of local pick-up by electrodes placed near the

From the Bird S. Coler Hospital Division of the Department of Medicine of the New York Medical College, New York City.
Received for publication Nov. 21, 1955.

heart, especially with regard to the S-T-T complex. In other words, juxtacardiac electrodes occasionally have a semidirect action.[3,4,6]

Another important consequence of vectorcardiography was the work of Burger[14] on the characteristics of the body as a volume conductor. He developed the basis for handling the realities of the volume conductor such as eccentricity, inhomogeneous conductivity, and finite size of the body. This also led to the detection of occasional instances of local pick-up of potentials by juxtacardiac electrodes.[7]

Several years ago we attempted to use vectorcardiographic leads in clinical electrocardiography.[2] We recorded sets of Leads I, aV_F, and V_2 or V_8; then we compared them to the usual 12-lead record. We found that the unipolar chest leads are indispensible and cannot be replaced by any single Z lead.* On the other hand, it became clear that only two limb leads are needed to supply all the information inherent in all the limb leads. Furthermore the two limb leads best suited for this turned out to be Leads I and aV_F.

METHOD

To put this conclusion to a formal test the following experiment was done. Electrocardiograms of three hundred patients were taken; each record consisted of two sets of leads. Set one consisted of only Leads I and aV_F; set two consisted of all six limb leads and the unipolar chest Leads V_2, V_3, and V_5. Each set was interpreted separately and then the interpretations were compared.

TABLE I. THE INCIDENCE THAT A GIVEN PATHOLOGIC ITEM WAS NOTED IN THE 300 ELECTROCARDIOGRAMS

Left ventricular preponderance or strain	85
Right ventricular preponderance or strain	6
Right bundle branch block	27
Left bundle branch block	21
Digitalis effects	74
A-V conduction disturbance	36
Extrasystoles	89
Auricular tachycardia or flutter	6
Auricular fibrillation	46
Myocardial infarction	
with anterior component	36
with posterior component	2
with inferior component	29
with lateral component	19

Differences were analyzed to determine whether Leads I and aV_F failed to supply significant clinical information compared to the information supplied by all six limb leads. No chest leads were included in the first set because we wanted to see what information could be derived from I and aV_F only. It was expected that significant material present only in the chest leads would be missed. This

*X, Y, and Z are the dextrosinistral, craniocaudal, and the dorsoventral body axes. In accord with their lead lines, I is the X lead, aV_F the Y lead, and V_2 or V_8 the Z lead. See Reference 1.

was taken into consideration in evaluating the diagnostic efficiency of the first set.

The ages of the patients ranged from 35 to 95 years. Table I shows the incidence of the different pathologic findings noted in the three hundred records; many cases showed multiple pathologic items.

RESULTS

In no instance did Leads II, III, aV_R, and aV_L contribute any significant information beyond that furnished by I and aV_F.

DISCUSSION

How can the results be reconciled with the many reports which indicate the need of aV_R for the diagnosis of right ventricular hypertrophy, of aV_L for the diagnosis of lateral wall infarction, and for Lead III for the diagnosis of "posterior" wall infarction? Our series of three hundred cases does not contain enough instances of these pathologic states to prove our thesis. However, for the past two years we have been looking for cases which show the need for limb leads besides I and aV_F. None has been found. Furthermore, since a preliminary report was published in July, 1954,[2] no such case has been brought to our attention. In this problem the absence of controverting evidence constitutes significant support for our thesis.

In the light of modern concepts the results lose a great deal of their novelty; they become quite obvious. If unipolar limb leads acted in a semidirect manner, then aV_L would be useful in determining the presence of an infarct in the myocardium adjacent and facing it. It would thereby help to map out the extent of an infarct. Likewise aV_R would be of use in detecting the excessive potentials of the base of the right ventricle which lies nearest the right shoulder. Such mechanisms are incompatible with the present state of knowledge of the recording of the heart's electrical activity. It is quite definite that the limb leads always act in a totally indirect or distant manner. This applies to both unipolar and bipolar leads; in this sense all limb leads are equivalent. Any two limb leads can reveal, theoretically at least, all the information inherent in all six limb leads because any two can determine the frontal heart vector.

Empirically Leads I and aV_F constitute the best combination for clinical use. Lead I furnishes the X, and aV_F the Y component of the frontal vector directly. The latter can thus be plotted directly on a graph with the familiar rectangular X, Y axes. For clinical interpretation this can be done mentally without written calculation. The correlation with the vectorcardiogram is simple since the latter commonly employs the same leads to supply the X and Y components of the heart vector.

Their greatest usefulness lies in the fact that they are critical leads for evaluating the normality of the P, QRS, S-T, and T phases because they indicate normality by a positive deflection and abnormality by a negative deflection. This rule is valid only for screening purposes; it has proved useful in teaching students how to make a first approximation of normality when interpreting

the tracing. (When aV_F is not taken, then the sum of Leads II and III divided by 2 can be substituted). This rule is based on the empirical findings that the mean vectors for the P, QRS, S-T, and T phases normally lie between 0 and plus 90 degrees.[8-10] In this range all vectors inscribe a positive deflection in both leads. Outside the range a vector inscribes a negative wave in one or both leads. The eye can easily distinguish between plus and minus; it is much more difficult to estimate magnitudes, which would be necessary if other limb leads were used.

The use of aV_R and aV_L in clinical practice is a left-over from the days when they were treated as semidirect leads. Their apparent usefulness found its greatest support in the classical investigations of G. B. Myers and his associates, who correlated the electrocardiogram with the pathology. The correlation was so good that there was little reason to suspect that some of the basic premises were faulty. The significance of Myer's work is not diminished thereby. On the contrary a preliminary reappraisal using modern concepts shows an even better correlation than he noted. This applies especially to the problem of high anterolateral wall infarcts.[7]

Lead aV_R was found useful in the diagnosis of right ventricular hypertrophy in that it occasionally showed a prominent R wave when the right chest leads did not.[11] Right hypertrophy is manifested in the tracing partly as a shift of the QRS vectors to the right and anteriorly. The right shift can be found by using I and aV_F. Lead aV_R does not contribute additional information. Its prominent R wave duplicates the information supplied by the S in Lead I. It cannot be considered equivalent to the prominent R in right chest leads taken at heart level. The chest leads show the anterior shift of the vector; the limb leads show the rightward shift.

Lead aV_L is supposed to be needed for the diagnosis of lateral wall infarction. Myers[12] states that Lead I is not an adequate substitute for aV_L since Lead I revealed an initial upstroke in five of eleven patients with lateral infarction with a QR pattern in aV_L which was either diagnostic or strongly suggestive of infarction. In addition, an infarct pattern in aV_L has been used to furnish the indication to take high chest leads to prove the presence of a high anterolateral infarct.

It is well known that a large Q and a negative T may be seen in aV_L normally.[8] This is probably due to the fact that the effective axis of aV_L has a large Z or anteroposterior component.[7] Therefore, an infarct pattern in aV_L is not diagnostic unless supported by similar curves in either Lead I or the chest leads. Every case that we have seen where high chest leads furnished the chief evidence of an infarction had in addition some abnormality in either Lead I or the standard unipolar chest leads. In Lead I, for example, there was a small R complex with or without a minute Q; or there was a T in I that was lower than the T in aV_F; or there was a suspicious S-T segment in I. In the standard chest leads there was either some deviation from the normal QRS pattern or a suspicious S-T-T complex. These abnormalities furnished the indication for recording high chest leads; we have not found a tracing either in Myer's reports or anywhere else which showed aV_L to be the only lead revealing a diagnostic infarct pattern.

Lead III is not useful in detecting a "posterior" infarct since it normally may show a large Q and negative T waves. If Lead II is used to assist in the diagnosis, then this is equivalent to reading Lead aV_F since Lead II plus III divided by 2 equals aV_F. It is more reasonable to take aV_F and omit Leads II and III. We have not yet come across an instance where we missed significant information by doing so. Note the quotation marks qualifying the word posterior. The prevalent custom of describing an infarct as posterior when the primary pattern is found in Lead aV_F is not logical. This applies also to the term anterior infarct when Lead I shows the primary Q-S-T-T pattern.[5] The limb leads cannot theoretically register the anteroposterior component of the heart vector. Therefore, if the primary infarct pattern is seen in aV_F one should diagnose an inferior or diaphragmatic infarct; when it is present in Lead I it indicates a lateral infarct.

It is worthwhile to use the newer concepts in the diagnosis of infarct localization. Since the limb electrodes are always indirect in their action it is illogical to say, for example, that the posterobasal wall of the left ventricle transmits its potentials to the left arm and so an infarct in this region should be detected by aV_L. Even the precordial electrodes are for the most part indirect in their pick-up; only occasionally has it been possible to detect a local pick-up effect. It is not logical to map out the size of the infarct by exploring the body with unipolar leads.

What the record can indicate is that an infarct is present and that it is large enough to inscribe a classical pattern. In general the larger the infarct, the more prominent is the infarct pattern. Small infarcts usually show only S-T-T changes while larger ones show prominent Q changes. This is not, however, equivalent to mapping out an infarct with unipolar exploring electrodes. The record can also localize the center of the infarct. Thus a primary infarct pattern in Lead I indicates that the center has a lateral component; in aV_F it shows an inferior component; in the anterior chest leads it shows an interior component; the reciprocal pattern in the anterior chest leads indicates a true posterior component.

The advantages of such a localization lies in a better correlation with the pathologic and clinical states. Changes resulting from multiple infarctions are more easily understood and followed. Furthermore, if one diagnoses a true posterior wall infarct one is alerted to the possibility of an auricular infarction, arrhythmias, and syncope. A preliminary application of these concepts to the material reported by Myers[7] shows a better correlation between the autopsy findings and the electrocardiogram. Furthermore, the poor correlation seen at times between the electrokymogram and the electrocardiogram may be explained in part by the faulty electrocardiographic diagnosis. Especially the so-called posterior infarcts were often anteroinferior and therefore should not have been expected to be accompanied by abnormal pulsations of the posterior heart border.

There are several reasons why it is not possible to use a single Z lead to replace the standard series of precordial leads. By virtue of the latter's close proximity to the heart they have two important characteristics: first, their

signal/noise ratio* is very favorable; secondly, they occasionally act in a semidirect manner and record the potentials originating in the myocardium adjacent to the exploring electrode.

The high signal/noise ratio of the precordial electrodes enables one to detect small but very significant abnormalities of the initial portion of the QRS complex such as QRS forms or a diminution of the R wave from right to left. This is often obscured in vectorcardiography by the large center spot. This means that the latter method cannot diagnose anterior infarcts as efficiently.

The local pick-up effect has been detected by several observers.[3,4,6,7] It has always involved the S-T-T complex in cases of left ventricular disease and has considerable clinical significance. It would be missed by a distant electrode like V_8. The vectorcardiogram would not show it. This seems to be the explanation in part for the recent observation by Holzmann[13] where he detected anterior wall pathology by the usual precordial leads and the vectorcardiogram failed to show it. Some of the discrepancy may be caused by the difference in the effective axes of the precordial leads and the vector leads.[7]

Should one attempt to use a single sagittal lead to take the place of the precordial series of leads then difficulties soon appear. To use V_8 as the Z lead means that one gives up the very favorable signal/noise ratio and the local pick-up of the precordial leads. Should one attempt to use V_2 then the problem of accurate and reproducible placement becomes the limiting factor. The density of the isopotential lines on the anterior chest wall is so great that the slightest change in the position of the exploring electrode has a large influence on the effective axis of V_2 and therefore on the tracing. If one attempts to minimize this factor by placing several interconnected electrodes or a single large electrode over the precordium then the precordial character of the electrode is destroyed and one is left with basically a distant electrode with an indirect pick-up. At present it seems unlikely that it will ever be possible to replace the series of precordial leads with a single lead and still obtain the same clinical information.

The diagnostic efficiency of the electrocardiogram in inferior wall infarction is not good whether all six limb leads are taken or only I and aV_F. Nor is the diagnostic efficiency in posterior wall infarcts good. The latter deficiency has led to the use of esophageal and posterior chest leads based on the assumption that these leads are semidirect. The esophageal leads have been proved to be impractical and unreliable. As for the posterior chest leads, personal study over several years has failed to disclose a single instance where such leads constituted the chief evidence of a myocardial infarction. If an electrocardiogram will manifest a posterior infarct, it will do so either by extension through Lead aV_F or by reciprocity through the anterior chest leads.

During the preparation of this report, there appeared an article by Biber and Feldman[15] which is in almost complete accord with us. They tested the adequacy of a six-lead tracing which consisted of Leads I, aV_F, V_1, V_3, 4_4, and V_6. They could detect no loss of clinical information with such a record as compared to the twelve-lead record. We take in addition Leads V_2 and V_5,

*Signal consists of the heart's electrical activity. Noise is made up of muscle tremor, A-C interference, electrode-skin potentials, etc.

because, in our opinion, the complete set from V_1 through V_6 is easier to evaluate for serial changes and is also easier to interpret by vector analysis.

A suggested lead routine is: I, aV_F, V_1 through V_6; in addition, in special cases, right chest leads (V_{3R}, V_{4R}, and V_{5R}) to diagnose right ventricular hypertrophy; high anterior chest leads (V_2 to V_4 in the second or third intercostal spaces) to diagnose high infarctions. In cases of arrhythmia Leads aV_F and V_1 seem best suited to show the auricular activity. Long strips should be taken in these leads. Furthermore a tracing should be done during carotid pressure which inhibits in some cases ventricular activity by increasing A-V block; whereby the P (or F) waves stand out clearly.

CONCLUSIONS

Two leads, I and aV_F, give all the information inherent in all six limb leads; the other limb leads are not needed for clinical use. This is supported by present knowledge of the physiology of the recording of the heart's electrical activity, by empirical findings, and by a controlled clinical experiment. While theoretically any two limb leads could suffice, I and aV_F are the best, because they indicate directly the X and Y components of the heart vector, and because they are critical leads in that they indicate probable normality or abnormality of the P, QRS, S-T, and T phases by a positive or negative wave, respectively.

The chest Leads V_1 through V_6 cannot be replaced by a single lead, because no single lead can duplicate their reproducibility, their high signal/noise ratio, and their occasional, but clinically significant, instances of local pick-up.

REFERENCES

1. Report of Committee on Electrocardiography, American Heart Association: Recommendations for Standardization of Electrocardiographic and Vectorcardiographic Leads, Circulation **10**:564, 1954.
2. Schaffer, A. I.: Useful and Useless Leads in Electrocardiography, Current Medical Digest **21**:31, 1954.
3. Grant, R. P.: The Relationship of Unipolar Chest Leads to the Electrical Field of the Heart, Circulation **1**:878, 1950.
4. Grant, R. P., Estes, E. H., Jr., and Doyle, J. T.: Spatial Vector Electrocardiography, Circulation **3**:182, 1951.
5. Grant, R. P., and Estes, E. H.: The Interpretation of the Electrocardiogram by Vector Methods, Emory University School of Medicine, 1949.
6. Schalnt, R. C., Levine, H. D., and Bailey, C. B.: Isolated T-Wave Negativity in the Ischemic Phase of Myocardial Infarction in Man, Circulation **10**:829, 1954.
7. Schaffer, A. I.: The Body as a Volume Conductor in Electrocardiography, Am. Heart J. **51**:588, 1956.
8. Lepeschkin, E.: Modern Electrocardiography, Baltimore, 1951, Williams & Wilkins Company, Paragraphs 224, 273, 283, 285, 290.
9. Burch, G. E., Abildskov, J. A., and Cronvich, J. A.: Studies of the Spatial Vectorcardiogram in Normal Man, Circulation **7**:558, 1953.
10. Abildskov, J. A.: A Study of the Spatial Vectorcardiogram on Normal Subjects Over the Age of Forty Years, Circulation **12**:286, 1955.
11. Myers, G. B., Klein, H. A., and Stofer, B. E.: The Electrocardiographic Diagnosis of Right Ventricular Hypertrophy, Am. Heart J. **35**:1, 1948.
12. Myers, G. B., Klein, H. A., and Stofer, B. E.: VII. Correlation of Electrocardiographic and Pathologic Findings in Lateral Infarction, Am. Heart J. **37**:374, 1949.
13. Holzmann, M.: Experiences With Rudimentary Anterior Wall Infarction, Am. Heart J. **50**:407, 1955.
14. Burger, H. C., and van Milaan, J. B.: Heart Vector and Leads. I., II., III., Brit. Heart J. **8**:157, 1946; **9**:154, 1947; **10**:229, 1949.
15. Biber, D., and Feldman, F. H.: The Relative Usefulness of the Electrocardiographic Leads, J. Newark Beth Israel Hosp. **6**:435, 1955.

A STUDY OF ELECTROCARDIOGRAPHIC AND SPATIAL VECTORCARDIOGRAPHIC CHANGES IN BUNDLE BRANCH BLOCK BY MEANS OF THE RECONSTRUCTION METHOD

HIDEO TOYOSHIMA, YASUSHI MIZUNO,* MITSUO HATTORI,** AND YOSHIKO SARUHASHI*

NAGOYA, JAPAN

INTRODUCTION

IT IS well known that specific patterns[1-11] such as the common type, the rare type, and the usual and unusual types of Wilson block appear in the standard extremity lead electrocardiograms of bundle branch block. The common type is characterized by a wide-topped or bifid tall R followed by an inverted T in Lead I and deep wide S or QS followed by an upright T in Lead III; the rare type, by a small R, deep wide S followed by an upright T in Lead I, and a tall, wide R followed by an inverted T in Lead III; the usual type of Wilson block, by concordant QRS complexes, small or moderate R followed by upright T in Leads I, II, and III; and the unusual type of Wilson block, by a discordant QRS complex in Leads I and III, upright concordant T in the three leads, small R and shallow S in Lead I and deep S and small R′ in Lead III.

Of these four types, the common and the rare types have been well known for a long time[1-5,8] and they have remained important subjects for discussion as to which type belongs to which side block of the His bundle. Recently, however, it has been ascertained that the common type is due to left bundle branch block (L.B.B.B.) and the three other types to right bundle branch block (R.B.B.B.). This ascertainment is due mainly to the works of Wilson and co-workers.[4,6,7,9,12,13] The usual[7] and unusual[16] types of Wilson block were reported by Wilson and co-workers, and were found by them to belong also to R.B.B.B.[7,14] The appearance of such a configuration as the usual type of the Wilson block, has been known by Kraus and Nicolai[15] under the name of neurasthenic wave, but the reason for such a wave to appear has not been ascertained. In three types of ECG of R.B.B.B., the usual type of Wilson block[14] is acknowledged by many authors to appear when the right bundle is blocked.

However, the occurrence of the two types, the unusual type of Wilson block and the rare type, in R.B.B.B., is not necessarily established. For example, according to Kobayashi,[10] these two types do not signify an isolated R.B.B.B.,

From the Research Institute of Environmental Medicine, Nagoya University.
Received for publication Dec. 30, 1955.

*Research Fellow in Cardiology, First Department of Internal Medicine, Nagoya University School of Medicine.

**Research Fellow in Cardiology, Third Department of Internal Medicine, Nagoya University School of Medicine.

but a combined block of R.B.B.B. and left partial B.B.B. The rare type of B.B.B. was obtained only when both the right bundle and the left posterior branch were sectioned. In the isolated R.B.B.B., the electrocardiographic pattern was always the usual type of Wilson block.

The same facts have been reported also by Pick[16] and by Yater.[17] Though the appearance of these electrocardiographic changes has been widely known and the correlation between the electrocardiographic patterns and the location of involvement investigated by many workers,[12,13] the theoretical explanation for the appearance of such a specific pattern in B.B.B. has not been established. One of the reasons may be that the mechanism of generation of muscle fiber potential has not yet been elucidated.

But if there is a theory which can explain the ECG and spatial VCG changes without any contradiction, we can accept it as established. The Bipolarity Theory of Wilson[18,19] adopted most generally by many authors is an excellent one from this point of view so far as it is concerned with the QRS complex. But in regard to the T wave, there remain problems that have to be investigated. Admitting that we accept the Limited Theory of Lewis,[20] to say nothing of the Bipolarity Theory, the appearance of an equivalent double layer between the muscle region in rest and that in activation may be expected, and hence, the spread of this equivalent double layer in accordance with the front of the spreading activation. If we accept such a spread of the equivalent double layer in the excitation process of the ventricle, we can suppose that the potential change at a given lead-point is proportional to the solid angle subtended by the equivalent double layer produced by ventricular activation. Such a view has been adopted by Wilson[18] and many others,[21] and is not a new one.

But to our regret, the potential change used hitherto for the explanation of the changes in QRS complex is not one obtained by measurement of the true solid angle at that instant, but is a hypothetic one based on the above view. For proof of the correctness of the Bipolarity Theory of Wilson or the Limited Potential Theory of Lewis, it is necessary therefore to obtain the true solid angle for each moment by actually measuring the solid angle. From this point of view the authors began the reconstruction of ECG's and spatial VCG's by means of solid angle measurement on a plaster model[25-27] of the heart after some experience in reconstruction from a graphic heart on paper.[22-24] At the beginning,[25,26] the solid angle measurement was carried out only on the epicardial surface and not on the endocardial one, so the solid angle on the endocardial surface was obtained by means of the rule of three.

In this paper, solid angle measurements were carried out on both the epicardial and free and septal endocardial surfaces by making specific plaster models consisting of five blocks. As it has been ascertained that the electrocardiographic and spatial vectorcardiographic changes in various conditions of the heart can be explained rationally by this reconstruction method of the ECG and spatial VCG of Toyoshima, we investigated the relationships between electrocardiographic and spatial vectorcardiographic changes and the ventricular excitation process in order to establish a theoretical explanation for the electrocardiographic change in bundle branch block.

METHOD

A plaster model of the heart from a cadaver with no heart disease was made in order to measure the solid angle at a given lead-point subtended by a given area of epicardial, endocardial, or septal surface. In order to make possible the solid angle measurement on the free and septal endocardial surfaces, a special plaster model of decomposable type consisting of five plaster blocks was made. When the solid angle measurements were being carried out on the free endocardial surfaces, the epicardial blocks were put aside to expose the endocardial surfaces, and when on the septal surfaces, the epicardial and free endocardial blocks were put aside to expose the septal surfaces. All surfaces of the plaster model were divided into many small equal divisions by a circular stamp. The right septal surface was divided into 9 divisions, the left septal surface into 8, the free right endocardial surface into 13, the free left endocardial surface into 10, and the epicardial surface into 69 divisions. The positions of the many lead-points in various leads were indicated by white marks painted at the tips of the poles set up around the plaster model.

When a solid angle measurement was to be made, the positions of the plaster model and of the lead-points were restored to their natural positions corresponding to those in the cadaver before excision of the heart. The solid angle Ω_i at a given lead-point subtended by a given division S_i on any surface was obtained from equation (1) by measuring the distance (r_i) from that lead-point to the center of the division, and the angle θ_i between the normal line at that center and the line linking that center and the lead-point.

$$\Omega_i = \frac{S_i \cos \theta_i}{r_i^2} \qquad (1),$$

where S_i is the area of the concerned division. In this article, S_i was regarded as a constant for every division and treated as a unit area. Results of solid angle measurement were compiled in a table in such a form that one can obtain the potential value at any lead-point and the spatial vector manifested by polyography and by the cube system method of Grishman and associates when a given division is activated. If the solid angle subtended by each division is known, the solid angle Ω subtended by a given extent of epi- or endocardial surface or the potential caused by activation of that area can be obtained by algebraic summation of each value of the solid angle, as follows.

$$\Omega = \sum_{i=0}^{n} \Omega_i \ .$$

According to the principle of electricity, the total sum of solid angles at a given lead-point must be nearly equal to zero in this table, because the ventricle at rest is a closed surface of double layer. But, in practice, it does not nearly become zero. The primary cause for this lies in the values of the solid angles at the basal region of the septum and the free walls, where the error of the value measured is largest due to the sudden curvature of the ventricular surface in this region. So, the values have been corrected for these regions so as to make the total value zero. Since the table has been presented in the article by Mizuno,[27]

one of our co-workers, reproduction of it has been omitted here. If the ventricular activation process is known previously, the ECG and VCG can be reconstructed from this table by adding the values in the table algebraically following the activation process of the ventricle.

In this paper, the ECG in standard bipolar extremity leads and unipolar extremity and precordial leads, and spatial VCG's by polyography and by Grishman's cube system method were reconstructed by means above mentioned for various conditions of ventricular activation in isolated or combined bundle branch block, and the relationships between the electrocardiographic and vectorcardiographic changes and ventricular activation process were pursued.

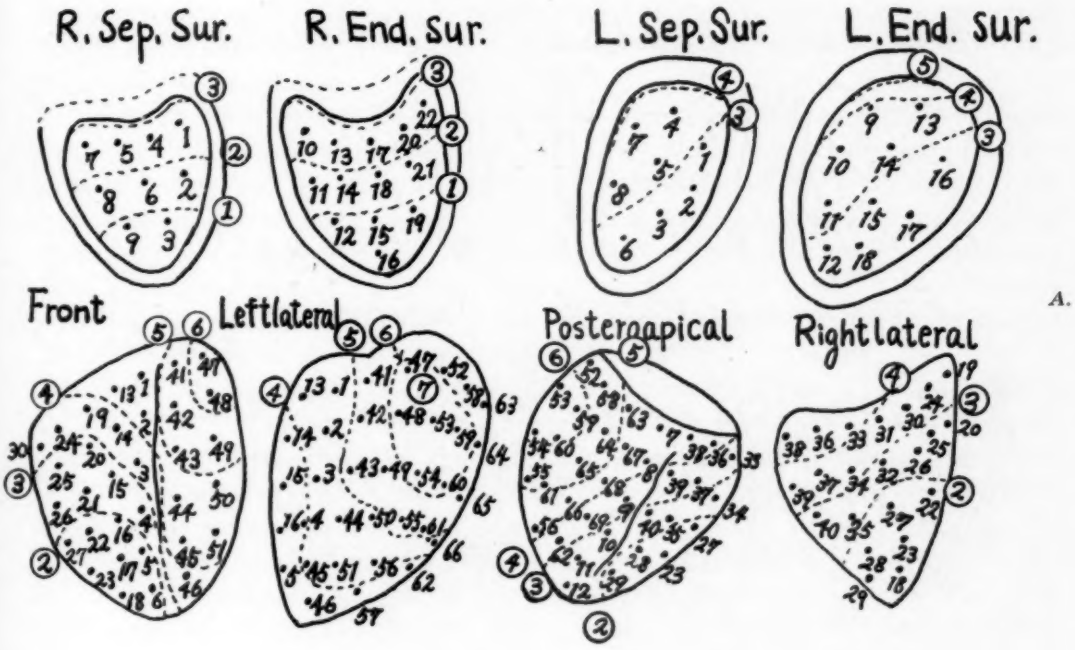
According to the studies of Sodi-Pallares and associates[29] and Prinzmetal and associates,[30] activation in the free ventricular wall of the blocked side ventricle spreads out in the normal manner from within to without. In experiments on the canine heart by the authors, the beginning of the intrinsic deflection of the direct lead ECG from the endocardial surface of the contralateral ventricular free wall was almost always earlier[31] than that from the opposite epicardial surface of that wall. Therefore, the reconstruction of ECG's and spatial VCG's in bundle branch block was carried out under the assumption that the activation process in the free wall of the blocked side ventricle may have been in the order of from endocardial surface to epicardial.

RESULTS

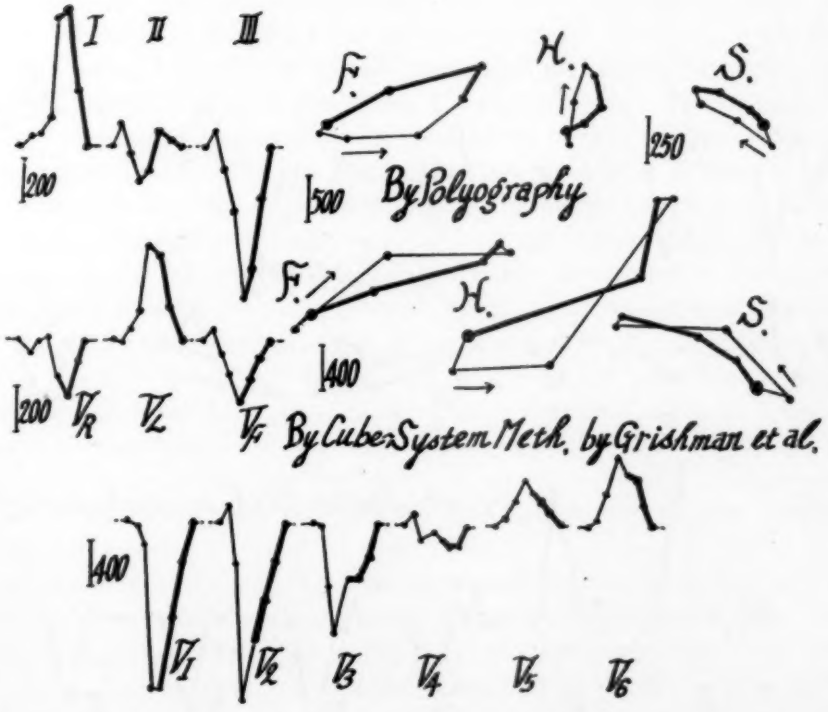
The contour maps in Fig. 1,*A* and Fig. 2,*A* are maps illustrating intraventricular activation processes in L.B.B.B. and R.B.B.B., obtained by the authors[32] from a comparative study of unipolar ECG's and spatial vectorcardiograms. For the reconstruction of ECG's and VCG's, it is necessary to obtain a map which makes it possible to understand the ventricular activation process as from a contour map. But, as there has been no investigation of such an idea, the activation processes in Figs. 1,*A* and 2,*A* were used in a trial reconstruction of ECG's and VCG's.

The numbered dotted lines show seriatim the front of spreading activation. Numbered points show the centers of each division of ventricular surfaces. Dotted lines and points in the upper row are those on the free endocardial and septal surfaces and those in the lower row, on the epicardial surface. In the activation process illustrated in Fig. 1, the activation began earliest in the subendocardial layer of the right ventricular apical aspect and then spread to the basal aspect. The activation in the left ventricle began two stages too late and spread from the posteroapical to the anterolateral. (One stage was 0.02 second and total QRS duration was 0.14 second.) The activation of the subepicardial muscle began earliest in the anteroapical aspect of the right ventricle and spread gradually to the basal aspect. The activation of the left ventricular subepicardial muscle began earlier in the posterior aspect than in the anterior aspect and ended in the anterolateral basal aspect.

In the activation process illustrated in Fig. 2, the process was more or less opposite in its activation order to that of L.B.B.B. The activation in the blocked



A.



B.

Fig. 1.—*A* illustrates a ventricular activation process in left bundle branch block obtained by comparative study of ventricular activation by means of unipolar lead ECG's and spatial VCG's. The contour maps of the upper row show the activation process of septal and free subendocardial surfaces and those of the lower row, the activation process of subepicardial surface. The activation begins earliest in the anteroapical aspect of the right ventricle and ends last in the anterobasal aspect of the left ventricle. *B* illustrates the ECG's and spatial VCG's reconstructed from the process in *A*.

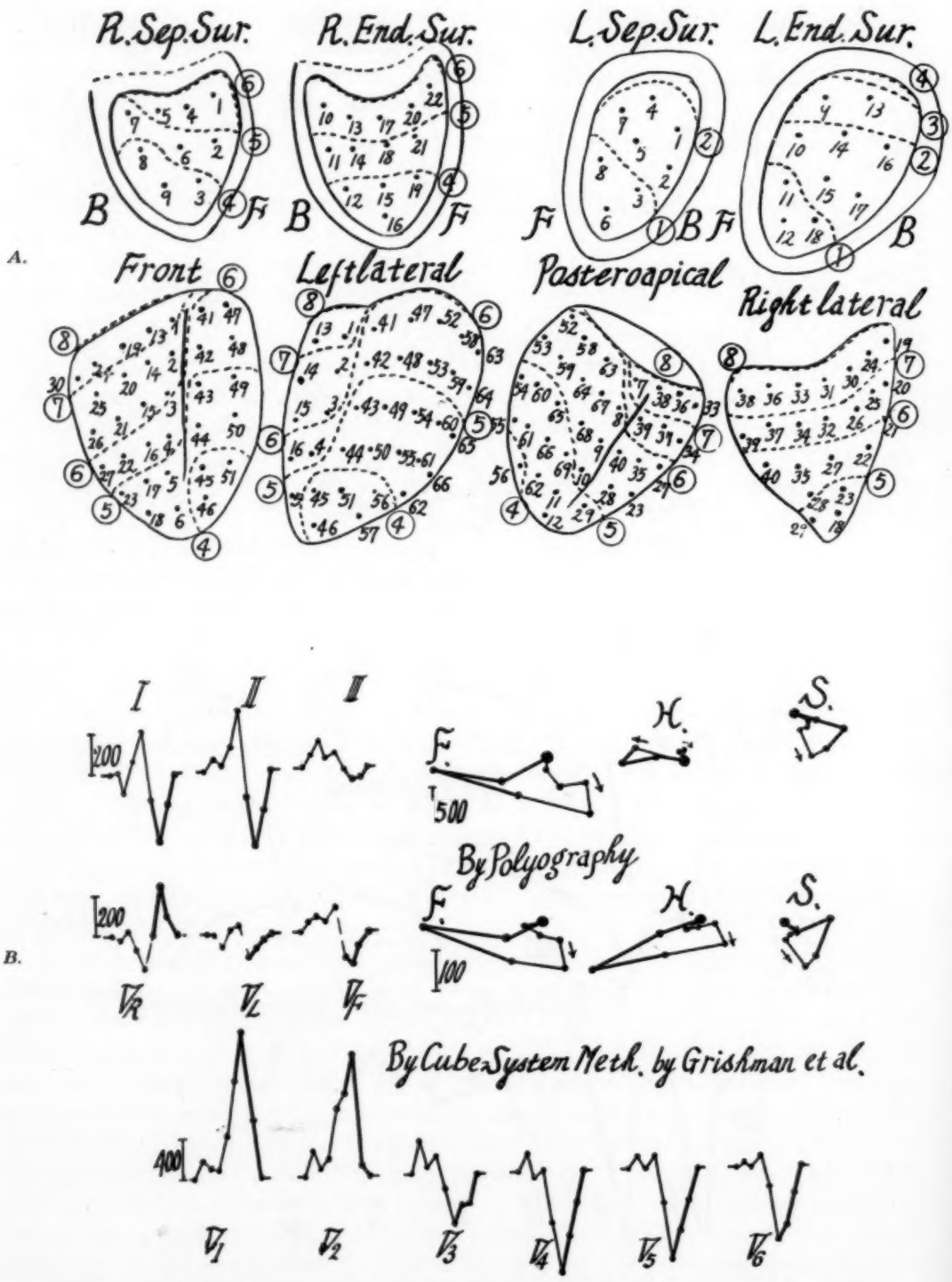


Fig. 2.—*A* illustrates a ventricular activation process in R.B.B.B. obtained in the same way as in Fig. 1. The activation begins earliest in the anteroapical aspect of the left ventricle and ends last in the right lateral anterobasal aspect of the right ventricle. *B* illustrates the ECG's and spatial VCG's reconstructed from the process in *A*.

side ventricle began earlier in the posterior aspect than in the anterior and ended in the anterolateral basal aspect. (One stage was 0.02 second and total QRS duration, 0.16 second.)

Tracings in Figs. 1,*B* and 2,*B* are the ECG's and VCG's reconstructed from the activation processes in Figs. 1,*A* and 2,*A*. I, II, and III are standard extremity leads; V_R, V_L, and V_F, unipolar extremity leads; and V_1 through V_6, standard unipolar precordial leads. In L.B.B.B. the ECG's display the common type with large R_I, deep S_{III} in the standard three leads, broad-topped and wide R in the left side precordial leads, and downward broad final deflection preceded by rather small thin R in the right side precordial leads. The spatial VCG is inscribed in the left posterior superior aspect.

In R.B.B.B. the ECG's display the usual type of Wilson block with broad shallow S waves in the standard three leads, tall R associated with delayed ventricular activation time in the right side precordial leads, and deep broad S in the left side precordial leads. The spatial VCG is inscribed in the right-anterior-inferior aspect.

Fig. 3,*A* is an example of a normal ventricular activation process producing the normal configurations of ECG's and spatial VCG's as seen in Fig. 3,*B*. ECG's and spatial VCG's display normal configuration with left axis deviation and semihorizontal position. Dotted lines in the ECG's and spatial VCG's are tracings reconstructed when the free endocardial surfaces of both sides were activated one stage earlier than the original process. Namely, in the first stage, both septal and endocardial surfaces of the apical region having been activated up to nearly the same level as in the case of the dotted lines. The tracings corrected by dotted line display a configuration resembling the normal more closely. Even with the heart in the same position, when the activation process is changed in such a manner that the depolarization of the right ventricular subepicardial surface begins earliest in the anterior aspect, nearer the base than that in Fig. 3,*A*, the pattern of the ECG's can be changed easily to the pattern of semivertical or vertical positions. According to our studies, a process such as that illustrated in Fig. 3,*A* is a most common type of ventricular activation process. If either branch of the His bundle is blocked, the muscle of the ventricle in the blocked side may be activated later than that in the intact side. So, to investigate the changes in configuration of the ECG's and spatial VCG's in blocks of either bundle, it might be reasonable to obtain ECG's and spatial VCG's reconstructed under the assumption that one side of the ventricular muscle was activated some stages later.

Tracings in Fig. 3,*C* are those reconstructed under the assumption that the left ventricular muscle is activated four stages later uniformly than the normal beginning in Fig. 3,*A*. Tracings in Fig. 3,*D* are those under the same assumption but for the right ventricle. Thick lines mean that the ECG and VCG of the practical lead may show some delay in this part of the tracing for abnormality of excitatory conduction in the blocked side ventricle. The ECG's in Fig. 3,*C* display the common type of B.B.B. with discordant QRS complex in standard extremity leads, deep QS or RS in right side precordial leads, and delay of the activation time in left side precordial leads. The long axis of the QRS spatial

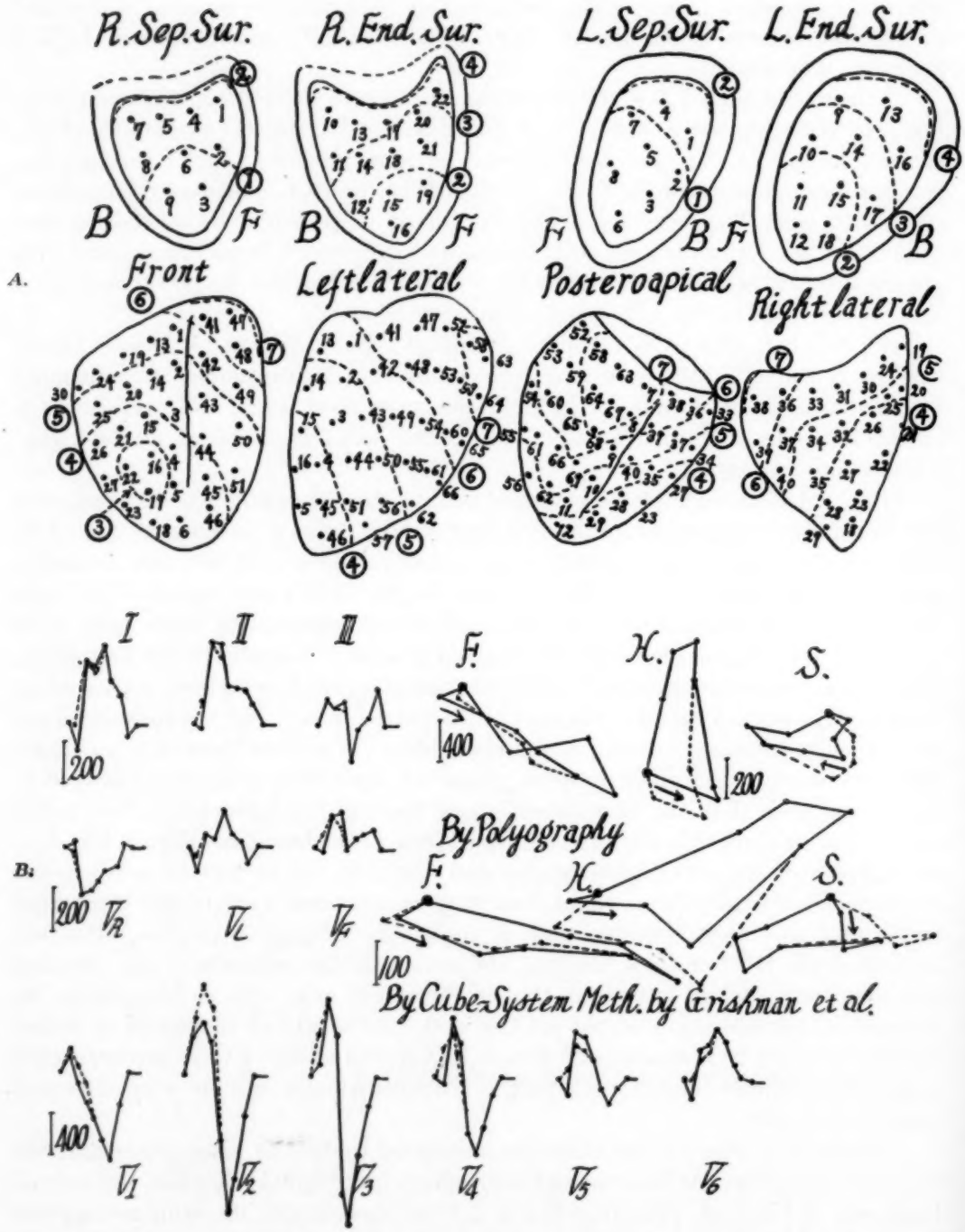


Fig. 3.—*A* illustrates an example of normal ventricular activation process producing normal ECG's and spatial VCG's. *B* illustrates the ECG's and spatial VCG's reconstructed from the activation process in *A*. Dotted lines in succeeding ECG's and spatial VCG's mean tracings obtained when activation of both side endocardial surfaces was fired one stage earlier than the activation process in *A*. *C* illustrates the ECG's and spatial VCG's reconstructed when the activation process in the left ventricle was delayed uniformly four stages later than that in *A*. *D* illustrates the ECG's and spatial VCG's reconstructed when the activation process in the right ventricle was delayed uniformly four stages later than that in *A*.

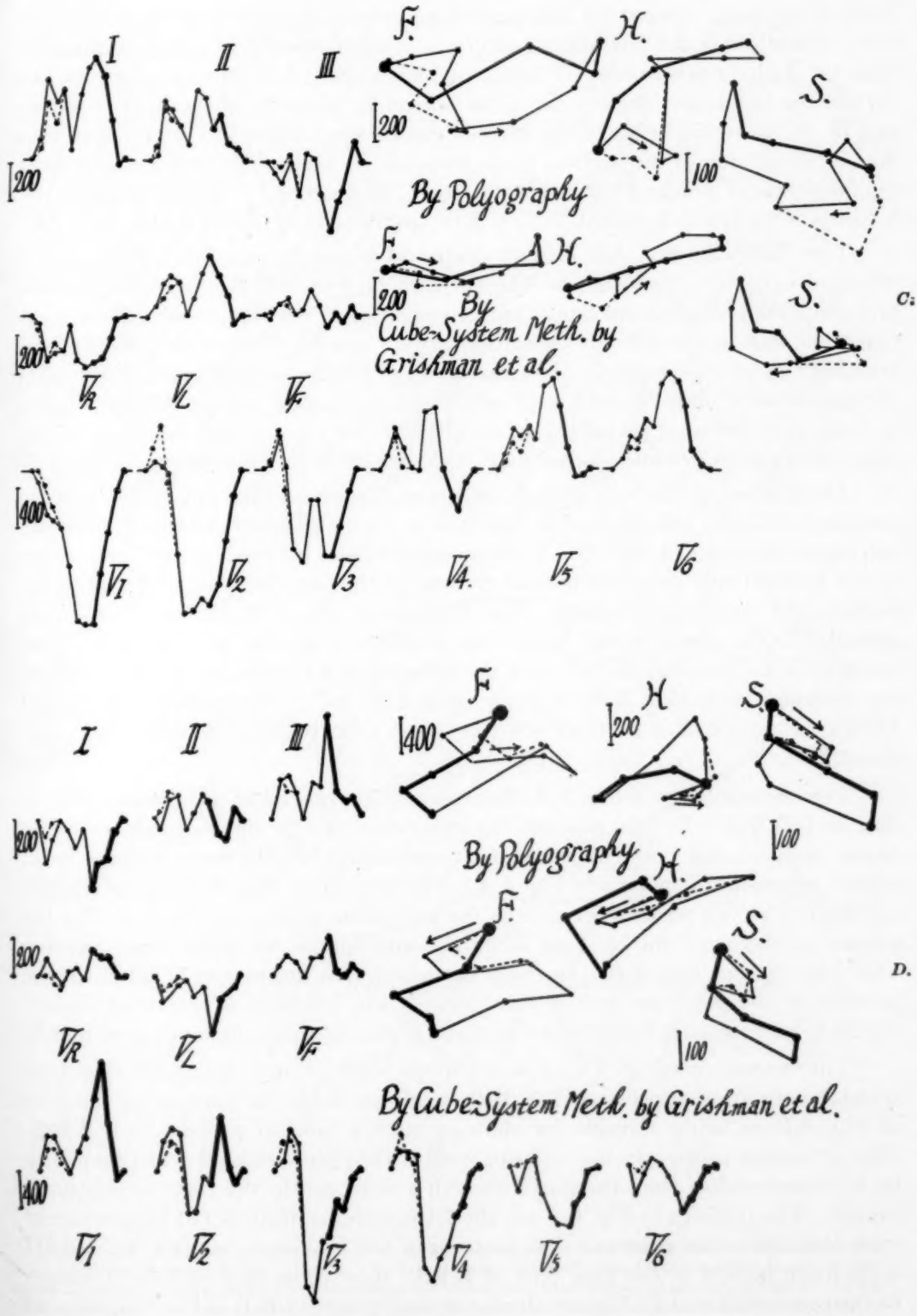


Fig. 3.—*C* and *D*. (*For legend see opposite page.*)

VCG is displaced toward the left-posterior-superior and the slow terminal portion is inscribed in the left upper posterior. The displacement is not so remarkable, for the long axis in normal conduction has already been displaced somewhat toward the left posterior because of the horizontal position of the heart. (Long axis of an anatomic heart of the plaster model was situated in such a position that its projection in the sagittal plane displays an inclination of 35 degrees with the longitudinal axis and in the frontal plane, 29 degrees.) But in general, the features of ECG's and spatial VCG's show specific characters of L.B.B.B.

The ECG's in Fig. 3,*D* display deep and broad S_I and tall late R_{III} in a standard extremity lead and are like the rare type of B.B.B. In the right side precordial lead we see a tall late R with conspicuously delayed ventricular activation time, and in the left side precordial lead, a smaller R wave and deeper and broader S wave than normal. The spatial VCG's were recorded mainly in right-anterior-inferior and the long axis was displaced to the right-anterior-inferior and the slow terminal portion was inscribed in the right-anterior-inferior. The features of the ECG's and spatial VCG's are like those in the rare type of B.B.B.

The contour maps in Fig. 4,*A* are maps illustrating the activation process modified from the process of the case in Fig. 3,*C* as suitably as possible for the activation process in L.B.B.B. The process has been planned so that activation in the blocked side ventricle is fired earliest in the anterior free wall and latest in the right anterobasal aspect. The tracings in Fig. 4,*B* are the ECG's and spatial VCG's reconstructed under the activation process of Fig. 4,*A*. The features of ECG's in I, II, III, and the precordial six leads are more typical of the common type of B.B.B. than those in Fig. 3,*C*. The features of spatial VCG's by Grishman's method are like those reported by Scherlis and associates.[35]

The contour maps in Fig. 5,*A* illustrate another process of ventricular activation in L.B.B.B. In this process, the activation of the blocked side ventricle began early in the posteroapical aspect and ended in the anterolateral basal aspect, opposite the process in Fig. 4,*A*. The tracings in Fig. 5,*B* are the ECG's and spatial VCG's reconstructed from the activation process in Fig. 5,*A*, but the general tendency of the changes in ECG's and spatial VCG's in this figure is very like that in Fig. 4,*B*. It must be noted that regardless of whether the anterior or the posterior wall of the blocked side ventricle is activated earlier, the ECG's and spatial VCG's show features typical of the common type of B.B.B.

The contour maps in Fig. 6,*A* are maps illustrating an example of a ventricular activation process of R.B.B.B. modified from the process in the case of Fig. 3,*D* as being suitable for showing such a kind of process in R.B.B.B. The activation process in the anterior wall of the right ventricle was planned to be activated earlier than the posterior wall and to end in the right lateral basal aspect. The tracings in Fig. 6,*B* are the ECG's and spatial VCG's reconstructed from this activation process. The features of the ECG's in Leads I, II, and III seem more typical of the rare type of B.B.B. than those in Fig. 3,*D*. Changes in the precordial lead ECG's are those commonly seen in R.B.B.B. The features of the spatial VCG's are very similar to that reported by Lasser and associates.[36]

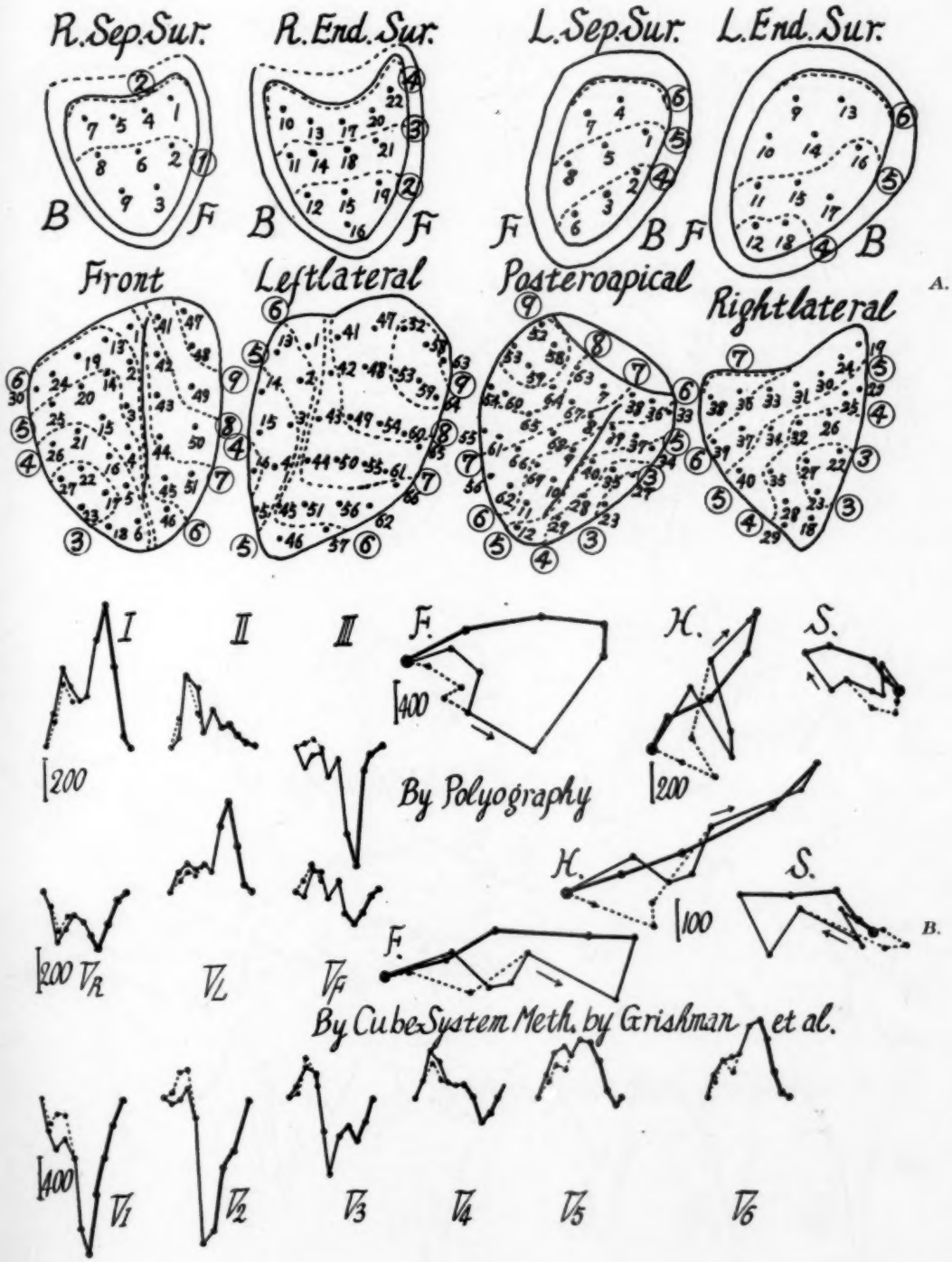


Fig. 4.—*A* illustrates ventricular activation process modified from the process in the case of Fig. 3,*C* as suitably as possible for L.B.B.B. In this process, the activation begins earliest in the anteroapical aspect of the right ventricle and ends in the left lateral basal aspect of the left ventricle. In the blocked side ventricle, the activation begins earlier in the anterior aspect than in the posterior. *B* illustrates the ECG's and spatial VCG's reconstructed from the activation process in *A*. The ECG's display the common type of B.B.B. See text.

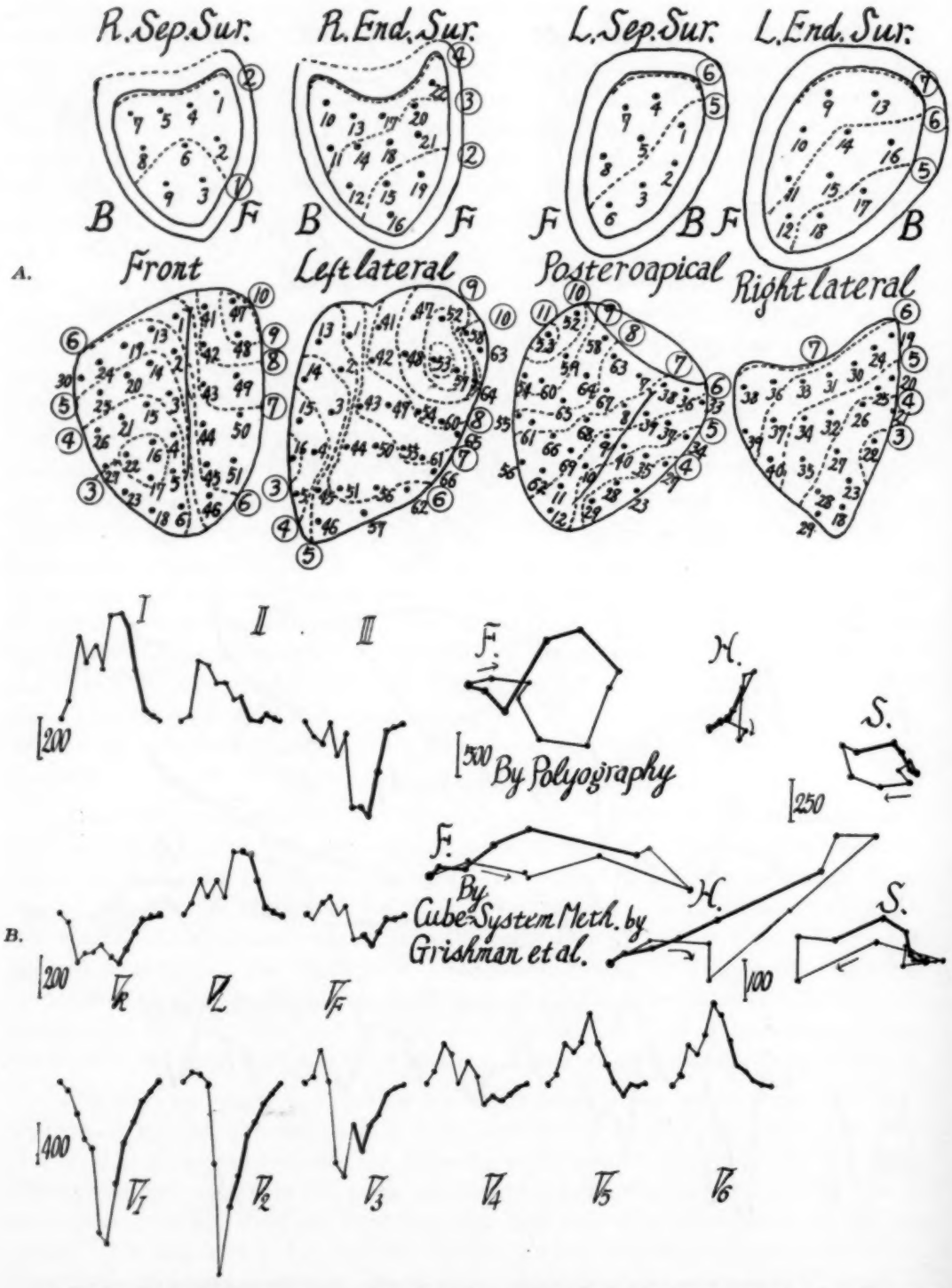


Fig. 5.—*A* illustrates another ventricular activation process of L.B.B.B. In this process, the activation begins earliest in the anteroapical aspect of the right ventricle and ends in the left lateral anterobasal aspect of the left ventricle. In the blocked side ventricle, activation begins earlier in the posterior aspect than in the anterior. *B* illustrates the ECG's and spatial VCG's reconstructed from the activation process in *A*. The ECG's display the common type of B.B.B. typical of L.B.B.B. See text.

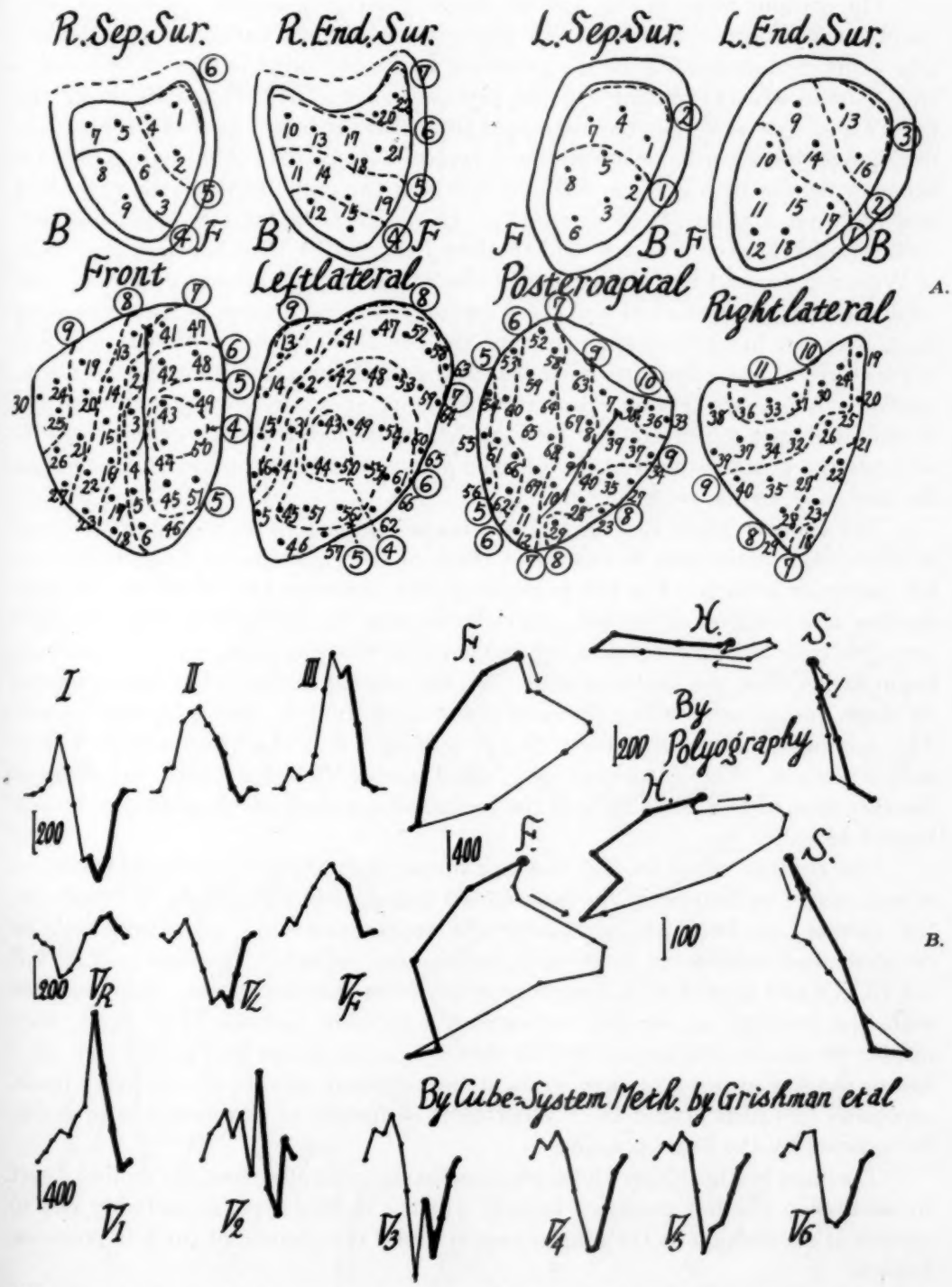


Fig. 6.—*A* illustrates the activation process modified from the process of the case in Fig. 3,*D* as suitably as possible for R.B.B.B. In this process, the activation begins earliest in the anterior aspect of the left ventricle and ends in the right lateral posterobasal aspect of the right ventricle. In the blocked side ventricle, activation begins earlier in the anterior aspect than in the posterior. *B* illustrates the ECG's and spatial VCG's reconstructed from the activation process in *A*. The ECG's display the rare type of B.B.B. in standard three leads and tall R with delayed activation time in V_1, V_2, and deep broad S in V_4, V_5, and V_6. See text.

The contour maps in Fig. 7,*A* are maps illustrating another process of ventricular activation in R.B.B.B. In this process, the activation of the blocked side ventricle began early in the posterior wall and ended in the right anterolateral basal aspect contrary to the process in Fig. 6,*A*. Fig. 7,*B* shows the ECG's and spatial VCG's reconstructed from this process. As seen in Fig. 7,*B*, the changes in precordial leads are those typical of R.B.B.B. The main difference between the features in Figs. 6,*B* and 7,*B* is in the magnitude of the preceding positive wave and late R in V_1 and V_2. In the ECG of the rare type, the preceding positive wave is higher and the late R is smaller than those in the ECG of Wilson type, and the diminution in size of the late R in the succeeding precordial lead is more marked and the appearance of the S wave in the succeeding leads is earlier in ECG of the rare type than in ECG of the Wilson type. But the features of the extremity leads are typical of the usual type of Wilson block, contrary to the rare type in Fig. 6,*B*. Therefore, it is noteworthy that both the Wilson and rare types are due to R.B.B.B. and that they are determined by whether the posterior wall of the blocked side ventricle is activated earlier than the posterior, or the reverse.

The contour maps in Fig. 8,*A* are maps illustrating an imaginary process of ventricular activation in combined block of the right bundle branch and the left posterior branch. For the presence of left posterior branch block, the conduction of excitation of the left ventricle through the posterior wall to the right ventricle may be markedly delayed and activation of the right anterior wall may begin earlier than the posterior wall. So, the process in Fig. 8 has been planned to show conspicuous delay of ventricular activation in the posterior aspect. The tracings in Fig. 8,*B* are the ECG's and spatial VCG's reconstructed from such a process. The features of ECG's and spatial VCG's are very like those of the rare type of B.B.B. in spite of the combined presence of left posterior bundle branch block.

The contour maps in Fig. 9,*A* are maps illustrating an imaginary process of ventricular activation in the isolated left posterior branch block. In this case, the process has been planned under the supposition that activation may be delayed most intensively in the left posterobasal aspect. Tracings in Fig. 9,*B* are ECG's and spatial VCG's reconstructed from such a process. As compared with the tracings in normal ventricular activation process (Fig. 3,*B*), they display no distinct difference, but as shown by a thick line in Fig. 9,*A*, we see a low or shallow appendant slow wave in the terminal portion of extremity leads, especially in Leads II and III. Whether it is upright or downward is probably determined by the heart position.

Tracings in Fig. 10 are those obtained experimentally from the canine heart by sectioning the left posterior branch. Delay in this type is probably due to abnormal conduction in the muscle region under the control of the left posterior branch.

DISCUSSION

There is no established theory on the genesis of the action current of a muscle fiber. But, according to various theories referred to most generally for an explanation of electrocardiograms, for example, the Bipolarity Theory of

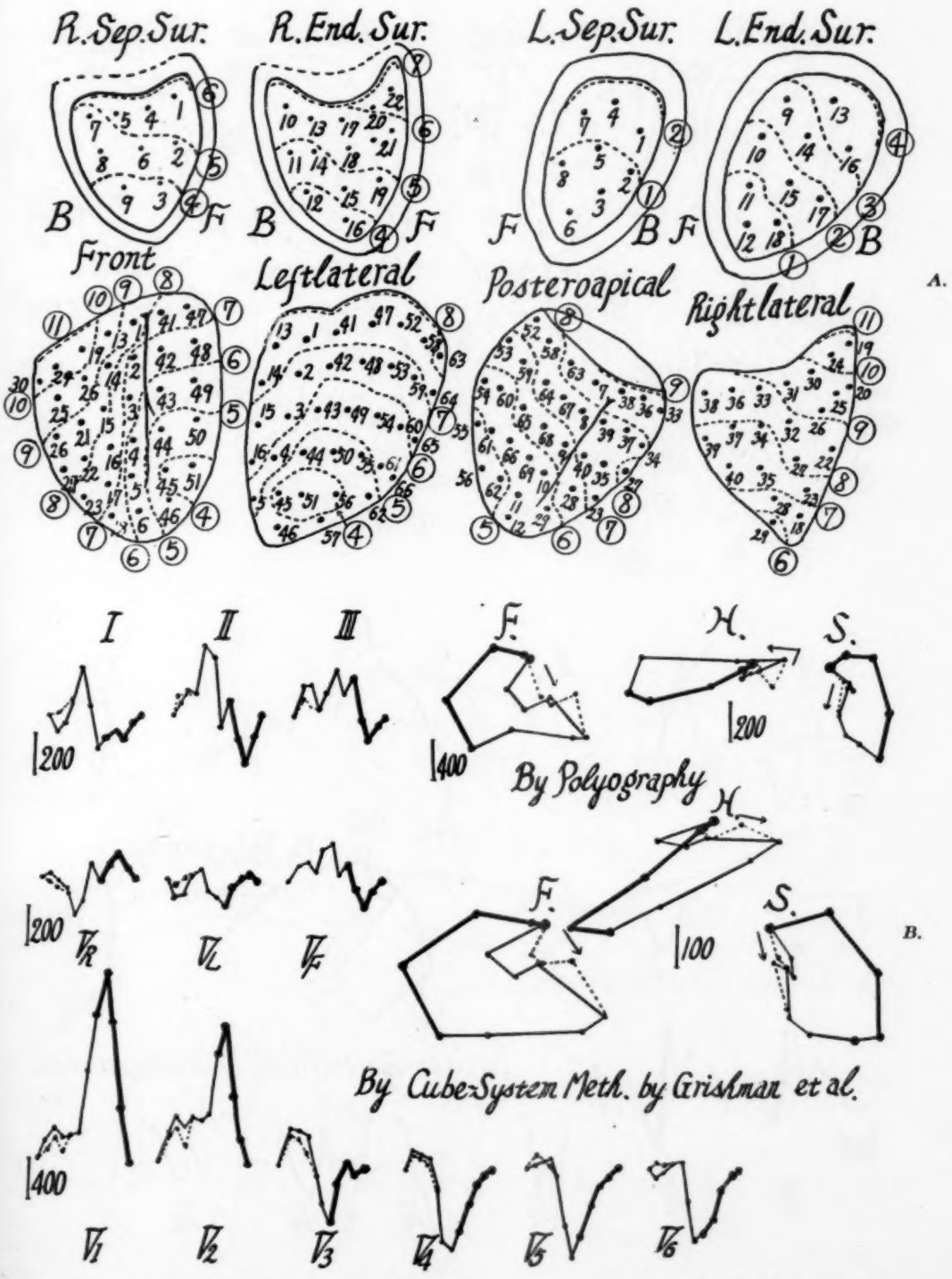


Fig. 7.—*A* illustrates another example of ventricular activation process of R.B.B.B. In this process, the activation begins earliest in the anteroapical aspect of the left ventricle and ends in the right lateral anterobasal aspect of the right ventricle. In the blocked side ventricle, the activation begins earlier in the posterior aspect than in the anterior. *B* illustrates the ECG's and spatial VCG's reconstructed from the activation process in *A*. The ECG's display the usual type of Wilson block in standard three leads. Precordial lead ECG's display tall R with delayed activation time in V_1 and V_2, and deep broad S in V_4.

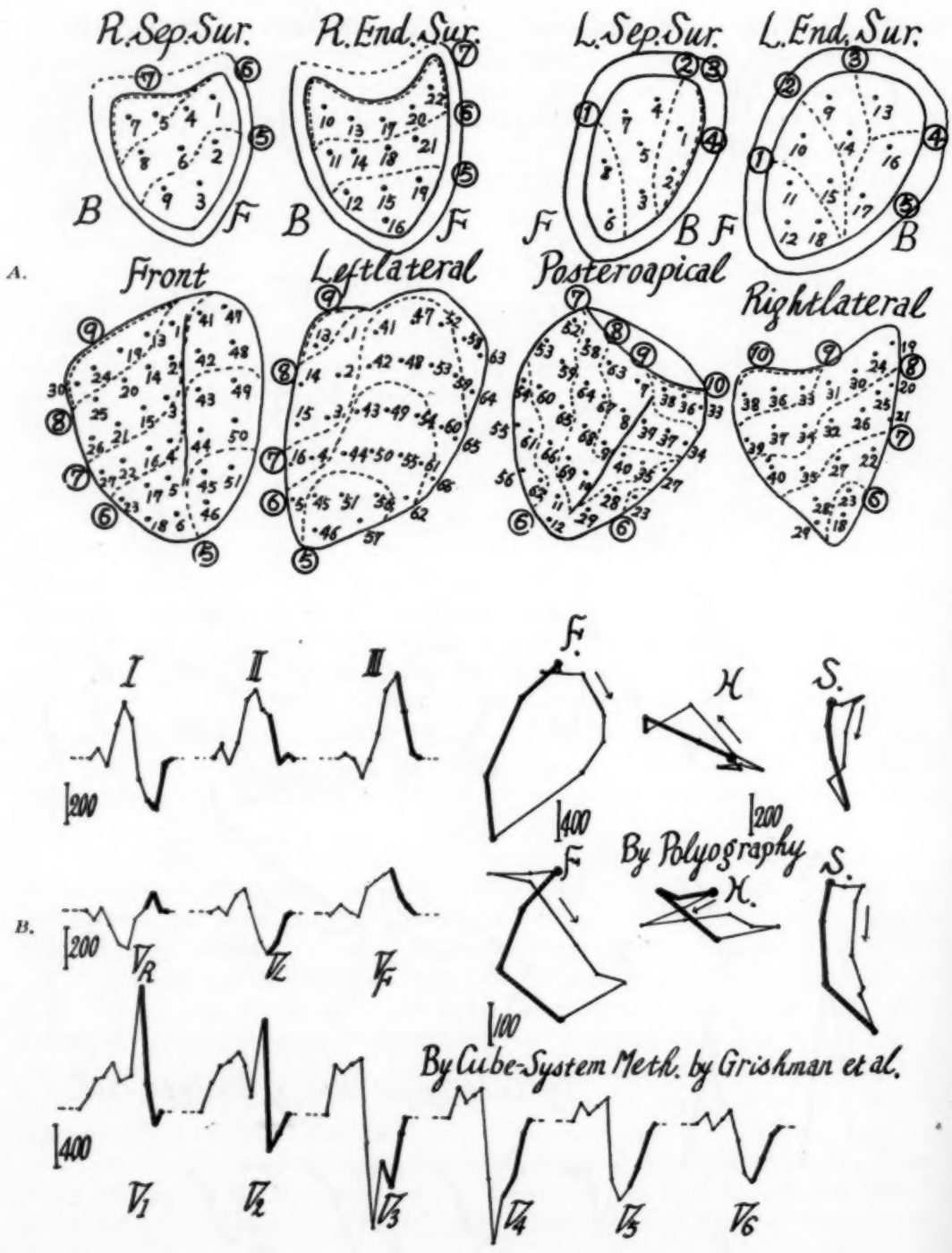


Fig. 8.—*A* illustrates a ventricular activation process of the combined block of the right bundle and left posterior branch. The activation begins earliest in the anteroapical aspect of the left ventricle and ends in the posterobasal aspect of the heart. In the right ventricle, the activation begins earlier in the anterior aspect than in the posterior. *B* illustrates the ECG's and spatial VCG's reconstructed from the process in *A*. The standard three extremity leads show ECG's typical of the rare type of B.B.B. V_1 and V_2 display tall R with delayed ventricular activation time and V_5 and V_6, deep broad S.

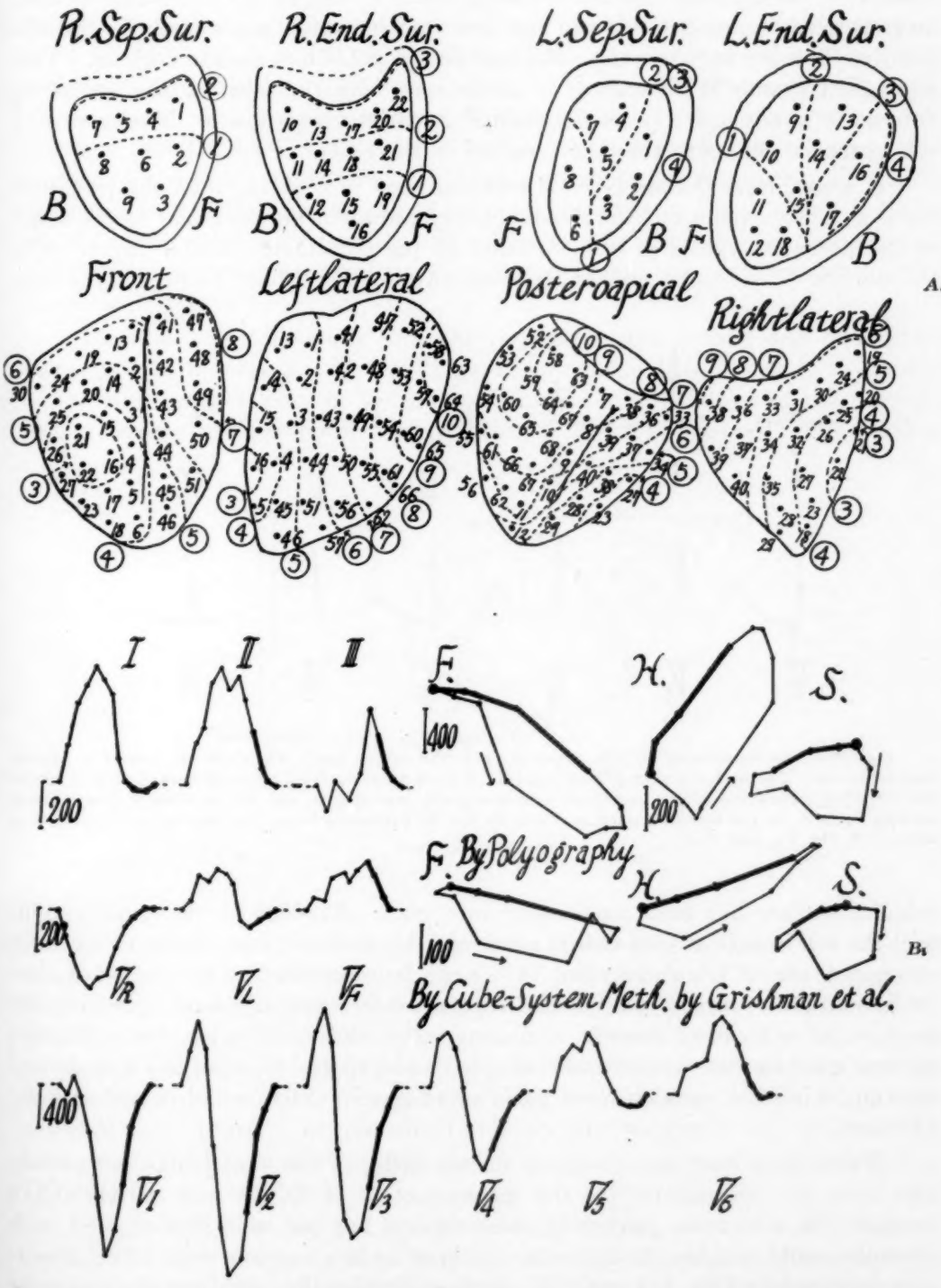


Fig. 9.—*A* **illustrates a ventricular activation process of the left posterior branch block. The activation of the subepicardial muscle begins earliest in the anterior aspect of the right ventricle as usual and ends in the posterobasal aspect of the left ventricle.** *B* **illustrates the ECG's and spatial VCG's reconstructed from the activation process in** *A*. **In the ECG appears, clinically, some delay at the portion shown by a thick line.**

Wilson or the Limited Theory of Lewis, we can imagine an equivalent double layer between a muscle region in rest and that in activation, namely in the Bipolarity Theory, between the polarized and depolarized muscle regions. This equivalent double layer spreads in accordance with the front of the spreading activation, producing a potential change at various lead-points. The record of this potential change is the QRS complex in the ECG.

If a sacciform double layer is given as seen in Fig. 11, then the potential value at a given point outside the double-layered sac is almost equal to the sum[35] of the potential values of each division of the partitioned double layer. The smaller the division, the smaller the error in potential value. Hence, if the ven-

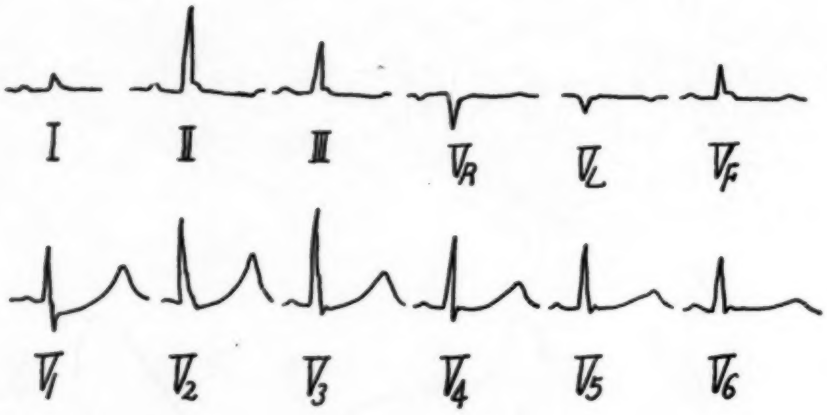


Fig. 10.—The reproduced ECG's obtained from the canine heart when the left posterior branch was sectioned. The features of precordial lead ECG's are different from those of human ECG's due to the different correlation of the heart and position of the lead-points, but an appendant slow wave is seen in V_1 to V_2 in the terminal portion of the ECG. In extremity leads, the slow appendant wave is seen in II, III, V_R, and V_F.

tricular surface has been partitioned into many divisions of the same extent, and the solid angle at lead-points subtended by each of these divisions has been measured, the ECG's and spatial VCG's can be reconstructed by means of algebraic summation of the solid angles subtended by these divisions following the ventricular activation process; and conversely, the validity of this activation process used for the reconstruction of ECG's and spatial VCG's can be evaluated by comparing the reconstructed ECG's and spatial VCG's with those actually obtained.

There have been many reports on the order of the ventricular activation; but they are not suitable for the reconstruction of ECG's and spatial VCG's because the activation process in these reports has not been investigated such that one could imagine the activation process, as in a contour map. The activation processes in Figs. 1,*A* and 2,*A* are those obtained by us[32] from the comparative study of unipolar lead ECG and spatial VCG. In these processes, most of the delay occurs in the transitional region between both sides of the free ventricular wall.

As shown in Figs. 1,*B* and 2,*B*, the ECG's and spatial VCG's reconstructed from these activation processes are strikingly similar to those actually obtained from patients with L.B.B.B. or R.B.B.B. Thus it may be reasonable to investigate the changes of the ECG's and spatial VCG's in bundle branch block by means of the reconstruction method of Toyoshima. This is also confirmed by the fact that the ECG's and spatial VCG's reconstructed from a normal ventricular activation process are very similar to those actually obtained from a normal person in the semihorizontal position, as is seen in Fig. 3,*A* and *B*. (The plaster model used in this article was somewhat in the horizontal position, contrary to the vertical position of the plaster model used for research on myocardial infarction reported by us.[26,36])

Therefore, it is believed that the reconstruction method will offer many important data necessary for the diagnosis of clinical B.B.B., for an evaluation of the Bipolarity Theory of Wilson, and for the establishment of the principle of bioelectric phenomena of the heart, if this method is applied with sound reason.

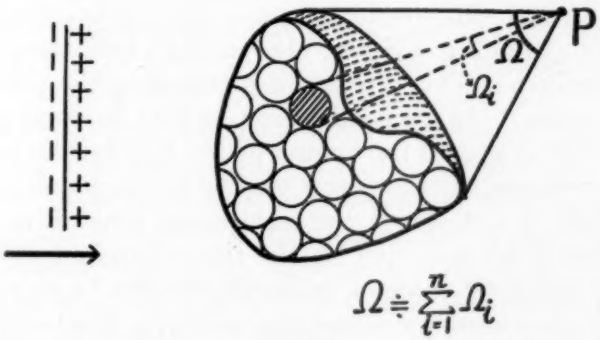


Fig. 11.—See text!

In bundle branch block, it is natural to suppose that the onset of activation would be delayed in the blocked side ventricle, and therefore it is not strange that the tracings reconstructed under such an assumption look very much like the ECG's and spatial VCG's actually obtained. Hence it may also be right to think that the features of ECG's and spatial VCG's (Figs. 4,*B* and 6,*B*), reconstructed from an imaginary ventricular activation process (Figs. 4,*A* and 6,*A*) modified suitably for a process which may actually happen in B.B.B., resemble more closely those of ECG's and spatial VCG's actually obtained than Fig. 3,*C* and *D*.

In L.B.B.B., whether the R is broad-topped, bifid, or double R in the precordial lead from points to the left of the transitional zone is greatly influenced by the degree of delay of activation of the left ventricle. The principal reason for the diminution of the initial R in the right side precordial leads, seen in L.B.B.B., is that the potential at this time is effected only by a small extent of the double layer produced in the left ventricular apical region. In the normal

heart without any B.B.B., the potential at this time is effected by an extensive double layer produced in both apical regions. The effects of the two double layers developed in both sides of the septum are cancelled and do not appear at this time.

In R.B.B.B., it is clear that the tall late R in V_1 and V_2 is due to the activation of the right ventricle, and the preceding R wave, to the left ventricle. When the anterior wall of the homolateral ventricle is activated earlier than the posterior, the late R in V_1 and V_2 is smaller than that obtained when the posterior wall is activated earlier, and vice versa. The S wave between the preceding R and late R in V_1 and V_2 may appear according to the grade of delay of the right ventricular activation. The earlier the onset of right ventricular activation, the deeper the S wave. The deep S wave in the left side precordial lead is somewhat deeper than that actually obtained. But such a deep S wave is seen often in experimental B.B.B. on the canine heart and in the human ECG of the vertical or semivertical position type. The presence of a deeper S wave in the semihorizontal position is a very rare occurrence.

As a reason for such a deep S wave, it may be supposed that the positions of the standard six precordial leads of this cadaver may have been in abnormal correlation to the heart (slightly higher than the normal level). The presence of a small S wave in V_5 in Fig. 3,*B* may also be a fact indicating the displacement of these six lead-points. (This is a problem regarding the position of the lead-point and heart shape.) As a second reason, it may be supposed that the ventricular activation process used for reconstruction may be different in some minor aspects from the real one. Perhaps this may be a very important reason.

As seen in Figs. 1, 2, 4, and 6, the transitional zone of the QRS complex in R.B.B.B. is displaced much farther to the right than in L.B.B.B., as reported by Wilson and associates.[37] One of the most effective factors for displacement of the transitional zone is an activation process in the blocked side ventricle and the grade of conduction delay. It is well known that the size of the QRS complex of ECG's in B.B.B. is larger in general than the normal. The reason for this has remained uncertain for a long time. Some have supposed that the normal ECG consists of two antagonistic electric forces due to the right and left ventricles; and hence, when either side of the His bundle is blocked, the ECG must become larger as a result of diminution of an antagonistic force of the other side ventricle. As seen in Figs. 5, 6, 9, etc., the ECG of B.B.B. has a possibility of becoming larger than normal to as much as 1.5 times normal size. So, it may be supposed that the abnormally large QRS complex in B.B.B. can be explained without any contradiction in the Bipolarity Theory, too.

A noteworthy fact is that in R.B.B.B. the features of the ECG's are those of the rare type of B.B.B. when the anterior wall is affected earlier by activation than the posterior wall in the block side ventricle (Fig. 6,*B*); and on the contrary, they are those of the usual type of Wilson block when the posterior wall is affected earlier by activation than the anterior (Fig. 7,*B*). But in L.B.B.B., the features of the ECG's always are those of the common type of B.B.B. when the anterior wall is activated earlier than the posterior wall or reversed in the blocked side ventricle (Figs. 4,*B* and 5,*B*). From these findings it was made clear why the features of clinical ECG's in L.B.B.B. always are those of the common type

of B.B.B., while in R.B.B.B. they show, at various times, the rare type and the usual and unusual types of Wilson block.

It has been well known in bundle branch block that there are two types of ECG, the common type and the rare type. But for a long time it has remained uncertain which type appears in which side of bundle branch block. However, the distinction that the common type of B.B.B. is due to L.B.B.B. and the rare type to R.B.B.B. was made clear by vigorous investigations by Wilson and his co-workers.[4,6,7,9,12,13] They reported also the specific pattern of so-called Wilson block[7] and the unusual type[14] of Wilson block, and stated that these two types belonged also to R.B.B.B. Later, this statement was acknowledged by many research workers. At present, the appearance of the common type of B.B.B. has been ascribed to L.B.B.B. and that of the rare type and the usual and unusual types of Wilson block, to R.B.B.B.

But the reasons why such various types of B.B.B. appear in R.B.B.B. in spite of the appearance of only a single pattern in L.B.B.B. remain uncertain, and there are many problems to be discussed concerning whether the various types of ECG in R.B.B.B. should be ascribed only to the isolated R.B.B.B. or to a combination of R.B.B.B. with a left partial block. The electrocardiographic changes in Figs. 4,*B*, 5,*B*, 6,*B*, and 7,*B* are very important from this point of view. As may be seen in these figures the features of ECG's in L.B.B.B. are always the common type whether the anterior or posterior wall is affected earlier by activation. But, the fact that in R.B.B.B. the electrocardiographic pattern shows two different types of the rare and usual types of Wilson block, according to whether the anterior wall is activated earlier than the posterior, or the reverse, is suggestive of the possibility of the appearance of two or more different types of pattern in standard extremity lead ECG's in R.B.B.B., when the effect of position of the heart and complexity of the block as in combination with left partial block are taken into account.

In the standard six precordial leads, however, the features of the ECG's in R.B.B.B. were nearly the same regardless of which wall was activated earlier. This fact indicates that for an evaluation of left or right B.B.B. it is advisable to consider the features of the standard precordial lead ECG's as was recommended by Wilson[37] and others.[42] Whatever features may appear in the standard extremity lead ECG's, the prolongation of the QRS duration to over 0.12 second, the tall R or late R accompanying the delay of activation time in the right side precordial leads, and the broad deep or shallow S wave in left side precordial leads are important criteria in R.B.B.B. The broad S wave in the left side precordial leads may be often obscured.

The articles on investigations as to which wall, anterior or posterior, may be affected earlier are few and far between. According to Wener and associates,[38] in left bundle branch block, the diaphragmatic and posterior surfaces of the left ventricle were consistently activated 0.01 to 0.08 second earlier than the anterolateral surface of the left ventricle, while in twenty normal persons the posterobasal surface of the left ventricle was activated 0.01 to 0.03 second later than the anterolateral surface of the left ventricle. As an explanation of this, he stated that earlier activation of the left posterobasal surface in L.B.B.B. may have been due

to the anatomic location of the conduction system in the posterobasal portion of the septum and, hence, the time required for the excitation process to spread from the right side of the septum to the posterior aspect of the left ventricle was shorter than to the anterolateral aspect.

But it is not so important in L.B.B.B. whether the anterior wall of the homolateral ventricle may be activated earlier or not, because the electrocardiographic patterns in the standard extremity leads and precordial leads in L.B.B.B. are almost the same whichever wall may be affected earlier.

On the other hand, it is a matter of importance in the isolated R.B.B.B. to know whether the posterior wall is activated later than the anterior wall or not. In R.B.B.B., when a block of the left posterior branch is combined, it is quite natural to expect delay in activation of the posterior wall of the right ventricle due to the disturbances in conduction of the left ventricular activation through the posterior wall to the right, and hence, to expect the appearance of the rare type of B.B.B. If the posterior wall should be activated later than the anterior in the isolated R.B.B.B., it is very difficult to determine by what cause this type of B.B.B. appears.

According to the results of experiments by Kobayashi,[10] the occurrence of the usual type of Wilson block was observed when the right bundle branch was sectioned; and the rare type of B.B.B. was observed only when a left posterior branch block was present in combination with R.B.B.B. Pick[16] and Yater[17] said also from the pathologic findings that the usual type of Wilson block resulted from isolated R.B.B.B., and the rare type of B.B.B. from a combined right B.B.B. and left partial block. Kato,[39] one of our associates, reported that in the common type and atypical (or Wilson) type of B.B.B. the posterior wall of the blocked side ventricle was always activated earlier than the anterior wall of the homolateral ventricle.

In our experiments on canine hearts, which will be reported in the near future by Hattori, one of our co-workers, the electrocardiographic pattern in standard extremity leads appeared also, always as the atypical (Wilson) type when the right bundle was sectioned without involving the left branch. From these findings, therefore, it is probably right to conclude that isolated R.B.B.B. produces only the atypical (or Wilson) type, and the rare type of B.B.B. appears when a left posterior branch block is present in combination with R.B.B.B. However the reason why the posterior wall of the right ventricle should be activated earlier than the anterior wall in R.B.B.B. remains uncertain.

Regarding the electrocardiographic changes of the isolated left posterior bundle branch block,[10] there has been no established theory. Hitherto, the changes of ECG's in bundle branch block have been discussed mainly in right and left B.B.B. and not in partial block of the left bundle, that is, in left posterior branch block or left anterior branch block. As compared with the relative smooth long course of the right bundle, the left bundle divides into two branches, the anterior and posterior branches, after a short course and it receives its blood supply from the different branches of the coronary artery. So, the probability of occurrence of partial blocking of the left bundle branch will not be so remote.

According to Kobayashi's experimental results on the canine heart, R_1 became small, R_2 and R_3 became large, S_2 and S_3 became small or disappeared, and QRS duration measured 0.06 to 0.08 second (normal duration 0.05 to 0.06 second) in his delta lead when the left posterior branch was sectioned. As shown by a thick line in Fig. 9,*B*, the appearance of the small appendant slow wave is expected in the last portion of the QRS complex in Leads II and III, and the occurrence of a marked delay may also be expected in this portion. In the right side precordial lead ECG, the occurrence of a gentle and delayed upstroke may be expected after the peak of the S wave and, sometimes, in the shape of a slow appendant wave. In the left side precordial lead the occurrence of the upright or downward slow appendant wave like that in Leads II or III may be expected.

Such a phenomenon as above mentioned can be recognized in the ECG's of left posterior branch block reproduced in the article of Kobayashi,[10] and it was also demonstrated by Hattori[40] similar to the ECG's reproduced in Fig. 10. The theoretical reasons for the appearance of the unusual type of Wilson block could not be ascertained yet in this article. Whether it is a phenomenon associated with isolated R.B.B.B. in abnormal heart position or a phenomenon resulting from combined R.B.B.B. and left partial block, still remains uncertain.

The Difference Between Hypertrophy and Bundle Branch Block.—In the heart with ventricular hypertrophy, it may be supposed that the ECG's and spatial VCG display similar changes to those in bundle branch block, because the activation in the subepicardial muscle layer is delayed due to the thickness of the hypertrophied ventricular wall. This is a matter well known in the clinical ECG from the fact that the ECG's in ventricular hypertrophy display prolongation of QRS duration to more than 0.1 second and a similar configuration to those of B.B.B. So the distinction between ventricular hypertrophy and B.B.B., especially incomplete B.B.B., is often very difficult, or almost impossible.

However from theoretical points of view it can be supposed that the key-point to differentiation of these two lies in the activation of the subendocardial muscle layer being delayed distinctly in the blocked side ventricle in spite of the beginning of subendocardial activation in the hypertrophied ventricle remaining in nearly normal condition. On the other hand, activation of the subepicardial muscle layer of the homolateral ventricle is delayed in both cases. The grade of delay, of course, differs according to the grade of hypertrophy and block. In B.B.B. of high grade, it is not difficult by the ECG to differentiate B.B.B. from ventricular hypertrophy because of the abnormally long duration of the QRS complex.

Therefore in B.B.B. of such a grade as requires differentiation from ventricular hypertrophy, the beginning of activation on the subepicardial muscle layer may be nearly equal to that in hypertrophy. Then, in B.B.B. with the same start of activation of subepicardial muscle layer as in ventricular hypertrophy, the difference in activation process in both cases lies only in the delay of activation of the blocked side subendocardial muscle layers. This finding has been established by esophageal lead ECG and catheterization. In other words, in left B.B.B., the cavity potential of the blocked side ventricle obtained directly

by means of catheterization[41] or semidirectly by an esophageal lead[38,42] at the atrial level displays early positivity, whereas the cavity potential is QS in configuration when the conduction is not impaired.

Fig. 12 illustrates the initial portion of the spatial VCG reconstructed under such an assumption. (ECG's are omitted.) The activation processes in the subepicardial muscle layers have been planned to be affected in the same time in both cases, but in the subendocardial muscle layer, to be affected two

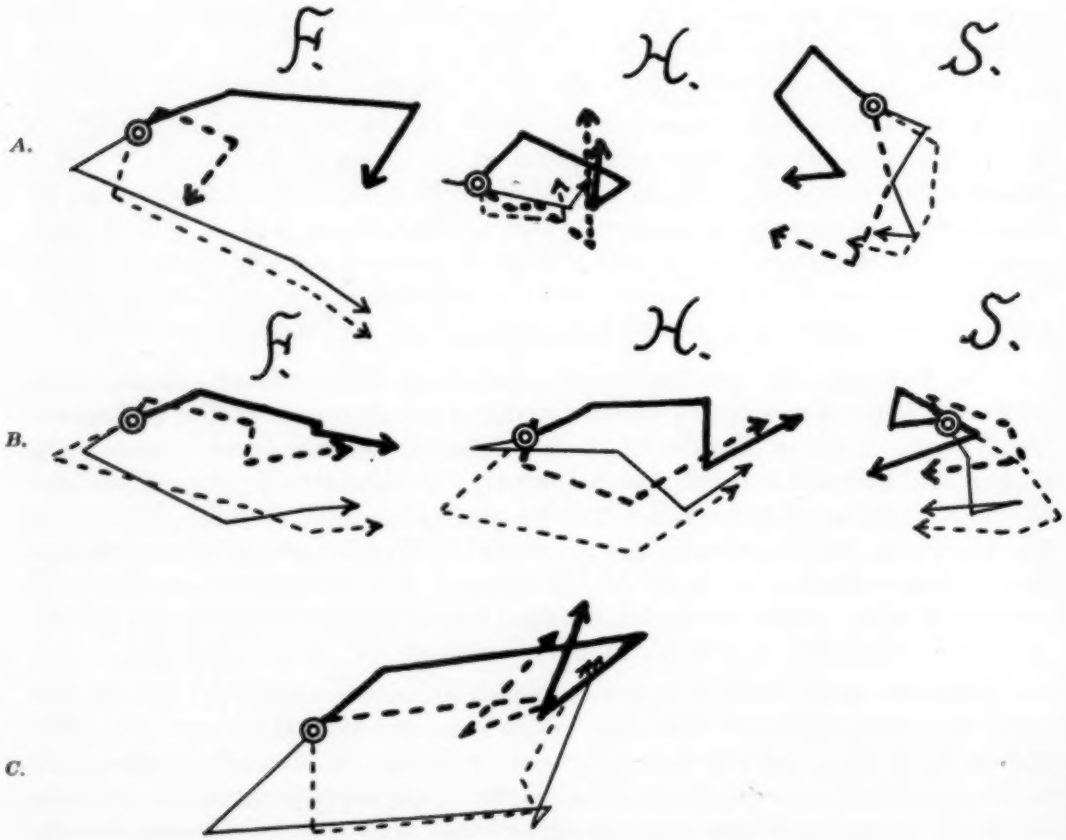


Fig. 12*A*.—Difference between the initial portions of the spatial VCG's of incomplete L.B.B.B. and left ventricular hypertrophy. Thick lines indicate initial portions of the spatial VCG's in L.B.B.B. and thin lines, those in left ventricular hypertrophy. They take directions opposite each other at the beginning. *A*, Polyography; *B*, Cube system method; *C*, Schellong's method.

stages late only in the blocked side ventricle. As seen in the spatial VCG's reconstructed, the initial direction of the spatial VCG is quite opposite in L.B.B.B. and left ventricular hypertrophy.

On the other hand, the initial directions of the spatial VCG were nearly the same in R.B.B.B. and in right ventricular hypertrophy. Hence the distinction of hypertrophy and B.B.B. by means of the spatial VCG would be relatively easy in the left ventricle from the findings expected theoretically; but, in practice, the diagnosis of hypertrophy or B.B.B. may not be necessarily as easy as seen in Fig. 12 because there are various types of configuration in normal

spatial VCG's according to the individual. If the spatial VCG in normal conditions is known previously, the diagnosis of such an incomplete B.B.B. will become more easy. Therefore the photographing of the spatial VCG in healthy condition is very desirable. In practice, the combined use of the spatial VCG, esophageal lead at atrial level, cavity lead, and standard precordial leads may be most desirable for a distinction between ventricular hypertrophy and incomplete B.B.B. But the distinct separation of B.B.B. and ventricular hypertrophy may be impossible in the final analysis, as Wilson has said.

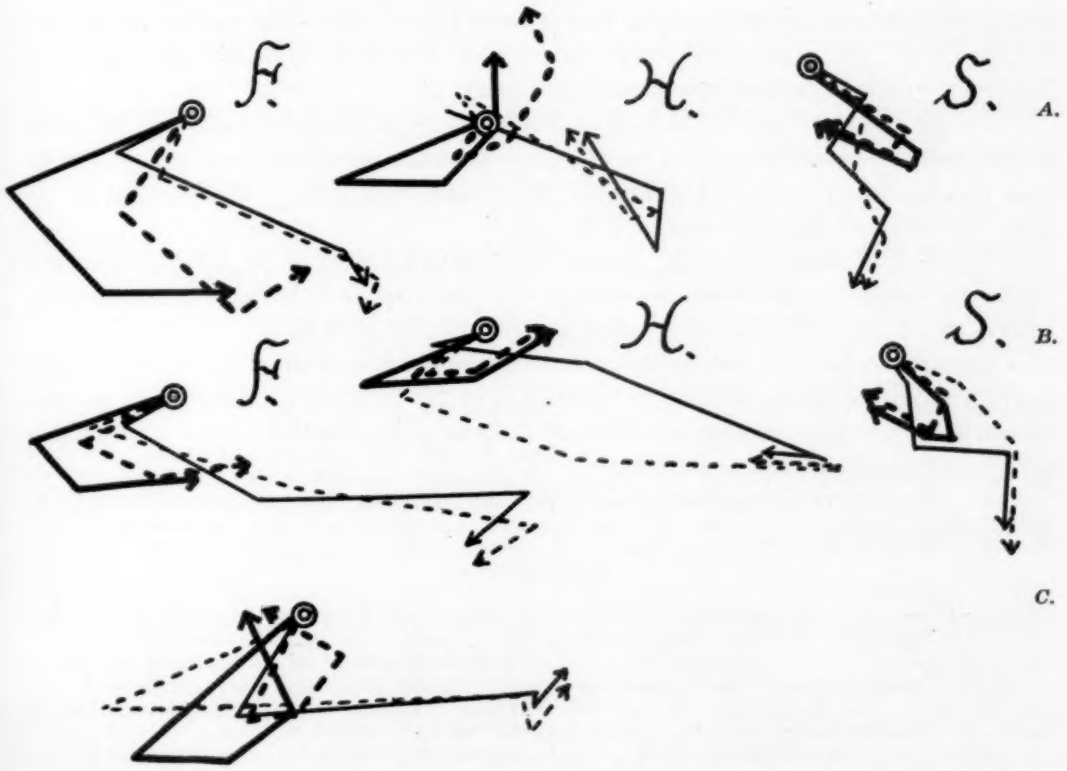


Fig. 12*B*.—Difference between the initial portions of the spatial VCG's in incomplete R.B.B.B. and right ventricular hypertrophy. Thick lines indicate initial portions of the spatial VCG's in R.B.B.B. and thin lines, those in right ventricular hypertrophy. They take a nearly similar direction at the beginning. *A*, Polyography; *B*, Cube system method; *C*, Schellong's method.

SUMMARY

1. The relationships between the electrocardiographic and spatial vectorcardiographic changes and ventricular activation process in the heart with bundle branch block were examined by means of the reconstruction of ECG's and spatial VCG's.

2. For the reconstruction of ECG's and spatial VCG's, a decomposable plaster model of the heart consisting of five blocks was used in order to make possible solid angle measurements on the free and septal endocardial surfaces.

3. In left bundle branch block, the features of the ECG in the standard three leads were always of the common type whichever wall, anterior or pos-

terior, of the blocked side ventricle was activated earlier; and the features of the six standard precordial leads were always ones typical of L.B.B.B. The spatial VCG was displaced to the left-posterior-superior, and slow terminal portion was inscribed in the left-posterior-superior aspect.

4. In R.B.B.B., the feature of the three standard extremity lead ECG's were of the usual type of Wilson block when the posterior wall of the blocked side ventricle was activated earlier than the homolateral anterior wall, and of the rare type of B.B.B. when the homolateral anterior wall was activated earlier than the homolateral posterior wall. The features of the standard six precordial lead ECG's were nearly the same in both cases. The spatial VCG was displaced to the right-anterior-posterior and the slow terminal portion was inscribed in the right-anterior-posterior aspect.

5. In the combined R.B.B.B. and left posterior branch block, the features of the three standard extremity lead ECG's were those of the rare type of B.B.B. and the features of the six standard precordial lead ECG's and spatial VCG were nearly the same as those in R.B.B.B.

6. If it is possible for the homolateral anterior wall to be activated earlier than the homolateral posterior wall in the isolated R.B.B.B., the appearance of the rare type of B.B.B. is possible in the isolated R.B.B.B.

7. In the isolated left posterior branch block, the three standard extremity lead ECG's were characterized by a slow appendant wave in the terminal portion of the QRS complex in Leads II, III, and often in I. In the spatial VCG, there was no particular characterization.

8. As criteria for diagnosis of right or left B.B.B., the precordial lead ECG is the most important.

REFERENCES

1. Eppinger, H., and Rothberger, C. J.: Zur Analyse des Electrocardiograms, Wien. klin. Wchnschr. **22**:1091, 1909.
2. Eppinger, H., and Rothberger, C. J.: Ueber die Folgen des Durchschneidung der Tawarascher Schenkel des Reizleitungs Systems, Ztschr. klin. Med. **70**:1, 1910.
3. Lewis, T., and Rothschild, M. A.: The Excitatory Process in the Dog's Heart, Part II. The Ventricles, Phil. Tr., Lond., Series B **206**:181, 1915.
4. Wilson, F. N., and Herrman, G. R.: An Experimental Study of Incomplete Bundle Branch Block and of the Refractory Period of the Heart of the Dog, Heart **8**:229, 1921.
5. Roberts, G. H., Crawford, J. H., Abramson, D. I., and Cardwell, J. C.: Experimental Bundle-Branch Block in the Cat, AM. HEART J. **7**:505, 1932.
6. Barker, P. S., Macleod, A. G., and Alexander, J.: The Excitatory Process Observed in the Exposed Human Heart, AM. HEART J. **5**:720, 1930.
7. Wilson, F. N., Johnston, F. D., Hill, I. G. W., Macleod, A. G., and Barker, P. S.: The Significance of Electrocardiograms Characterized by an Abnormally Long QRS Interval and by Broad S-Deflections in Lead I, AM. HEART J. **9**:459, 1934.
8. Bayley, R. H.: The Frequency and Significance of Right Bundle-Branch Block, Am. J. M. Sc. **188**:236, 1934.
9. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Order of Ventricular Excitation in Human Bundle-Branch Block, AM. HEART J. **7**:305, 1932.
10. Kobayashi, T.: Experimental Study of Bundle Branch Block, (In Jap.), J. Jap. Soc. Int. Med. **30**:1, 1942.
11. Kountz, W. B., Prinzmetal, M., Pearson, E. F., and Koenig, K. F.: The Effect of Position of the Heart on the Electrocardiogram. I. The Electrocardiogram in Revived Perfused Human Hearts in Normal Position, AM. HEART J. **10**:605, 1935.
12. Wilson, F. N., Macleod, M. A., and Barker, P. S.: The Interpretation of Initial Deflections of the Ventricular Complex of the ECG, AM. HEART J. **6**:637, 1931.
13. Wilson, F. N., Macleod, A. G., and Barker, P. S.: Order of Ventricular Excitation in Human Bundle-Branch Block, AM. HEART J. **7**:305, 1932.
14. Wilson, F. N., Johnston, F. D., and Barker, P. S.: Electrocardiograms of an Unusual Type in Right Bundle-Branch Block, AM. HEART J. **9**:472, 1934.

15. Kraus, F., and Nicolai, G.: Das EKG des Gesunden und Kranken Menschen, Leipzig, 1910.
16. Pick, A.: Beitrag zur Frage des Atypischen Schenkelblocks, Ztschr. klin. Med. **129**:719, 1933.
17. Yater, W. M.: Pathogenesis of the Bundle Branch Block; Review of the Literature; Report of Sixteen Cases With Necropsy and of Six Cases With Detailed Histologic Study of the Conduction System, Arch. Int. Med. **62**:1, 1938.
18. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Distribution of the Actioncurrent Produced by Heart Muscle and Other Excitable Tissue Immersed in Extensive Conducting Media, J. Gen. Physiol. **16**:423, 1933.
19. Wilson, F. N., Johnston, F. D., and Hill, I. G. W.: The Interpretation of the Galvanometric Curves Obtained When One Electrode Is Distant From the Heart and the Other Near or in Contact With the Ventricular Surface. Part II. Observations on the Mammalian Heart, AM. HEART J. **10**:176, 1934.
20. Lewis, T.: Interpretation of the Initial Phase of the Electrocardiogram With Special Reference to the Theory of "Limited Potential Differences," Arch. Int. Med. **30**:269, 1922.
21. Ashman, R.: Essentials of ECG, New York, 1942, The Macmillan Company.
22. Toyoshima, H., and Yamada, T.: Vectorial Study of the Bioelectric Phenomena of Heart by Means of Polyography, (In Jap.), Nippon J. Angiocardiol. **15**:127, 1951.
23. Yamada, T.: Local Diagnosis of Myocardial Injury by Vectorpolyography. Part I, Subepicardial Injury, Nippon J. Angiocardiol. **16**:41, 1951.
24. Yamada, T.: Local Diagnosis of Myocardial Injury by Vectorpolyography. Part II, Subendocardial and Subepicardial Injury, Nippon J. Angiocardiol. **16**:256, 1952.
25. Yamada, K.: Potential at Various Points to Lead Due to Activation of Ventricular Surface and Its Application to Clinical Electrocardiography and Vectorcardiography. Part I and Part II. (In Jap.) Jap. Circulation J. **18**:262 and 319, 1954.
26. Yamada, K.: Decision of Location and Extent of Myocardial Infarction by the Unipolar Lead Electrocardiography and Vectorcardiography, Nagoya J. M. Sc. **17**:322, 1954.
27. Mizuno, Y.: Researches for the Spatial Vectorcardiography and Electrocardiography by Means of the Reconstruction Method, Jap. Circulation J. **19**:397, 1955.
28. Matsunami, B.: Research for the Electrocardiographic and Vectorcardiographic Changes in the Ventricular Hypertrophy by Means of Reconstruction Method of ECG and VCG, Jap. Circulation J. **19**:387, 1955.
29. Sodi-Pallares, D., Bisteni, A., Medrano, G. A., and Cisneros, F.: The Activation of the Free Left Ventricular Wall in the Dog's Heart, AM. HEART J. **49**:587, 1955.
30. Kennamer, R., and Prinzmetal, M.: Depolarization of the Ventricle With Bundle Branch Block. Studies on the Mechanism of Ventricular Activity. X, AM. HEART J. **47**:769, 1954.
31. Toyoshima, H., Kato, R., and Mizuno, Y.: Research for the Intraventricular Activation Process by Means of VCG and ECG (3rd Report); On the Experimental Ventricular Premature Beat. (In Jap.), Published From Speech in 19th Ann. Meet. of Jap. Circulation Soc. in Kyoto, April, 1955.
32. Toyoshima, H.: Intraventricular Excitation Process and Electro- and Vectorcardiogram (In Jap.), Saishin Igaku **9**:1442, 1954.
33. Scherlis, L., and Grishman, A.: Spatial Vectorcardiography: Left Bundle Branch Block and Left Ventricular Hypertrophy. II, AM. HEART J. **41**:494, 1951.
34. Lasser, R. P., and Grishman, A.: Spatial Vectorcardiography: Right Bundle Branch Block. VIII, AM. HEART J. **42**:513, 1951.
35. Toyoshima, H., Otani, K., and Yamada, T.: Electrical Study of So-Called Electrical Heart Axis in Vectorcardiography, Part II, Nippon J. Angiocardiol. **16**:323, 1953.
36. Toyoshima, H., and Yamada, K.: Potential at Various Points to Lead Due to Activation Process of Ventricular Surface (In Jap.), Jap. Circulation J. **17**:218, 1953.
37. Wilson, F. N., Rosenbaum, E. F., and Johnston, F. D.: Interpretation of the Ventricular Complex of the ECG, Adv. Int. Med. **2**:1, 1947.
38. Wener, J., Scherlis, L., and Sandberg, A. A.: The Spread of the Excitatory Process and the Left Ventricular Cavity Potentials in Left Bundle Branch Block as Studied With Esophageal Leads, AM. HEART J. **41**:864, 1951.
39. Kato, R.: Analysis of the Ventricular Activation Process From the Vectorpolyogram and Unipolar ECG; On the Normals, Ventricular Hypertrophies, Bundle Branch Blocks and Ventricular Premature Beat (In Jap.), Jap. Circulation J. **19**:287, 1955.
40. Toyoshima, H., and Hattori, M.: Experimental Studies on the Electrocardiographic and Vectorcardiographic Change in Bundle Branch Block (In Jap.), Published From Speech in 19th Ann. Meet. of Jap. Circulation Soc. in Kyoto, April, 1955.
41. Sodi-Pallares, D., Estandía, A., Soberón, J., and Rodríguez, M. I.: The Left Intraventricular Potential of the Human Heart. II. Criteria for Diagnosis of Incomplete Bundle Branch Block, AM. HEART J. **40**:655, 1950.
42. Wener, J., Sandberg, M. D., and Scherlis, L.: The Nature of the RS-T Segment Displacement as Studied With Esophageal Leads, AM. HEART J. **41**:410, 1951.
43. Richman, J. L., and Wolff, L.: Left Bundle Branch Block Masquerading as Right Bundle Branch Block, AM. HEART J. **47**:383, 1954.

THE VENTRICULAR PATTERNS IN THE RIGHT PRECORDIAL LEADS IN MITRAL STENOSIS

THOMAS L. MORRIS, M.B., B.CH., AND WILLIAM WHITAKER, M.D., M.R.C.P.

SHEFFIELD, ENGLAND

RIGHT ventricular hypertrophy in patients with mitral stenosis is not associated with a constant electrocardiographic pattern. Wilson and associates[1] studied precordial electrocardiograms in patients with right ventricular hypertrophy and regarded a tall R wave, a prominent q wave, and inverted T wave in Lead V_1, together with a small r and a deep S wave in Lead V_5 or V_6 as characteristic of right ventricular hypertrophy. Myers and associates[2] agreed with Wilson in the necessity of employing multiple precordial leads for estimating right ventricular hypertrophy and mentioned that similar electrocardiographic patterns resulted whatever the cause of right ventricular hypertrophy. They described increased amplitude and duration of the R wave with delay in onset of its peak and absence of the S deflection in Lead V_1, and a deep S wave in V_6, due to prolonged activation of the right ventricle, as the classical pattern of right ventricular hypertrophy. However, Kossmann and associates[3] found that less than one third of patients with right ventricular hypertrophy secondary to mitral stenosis had a late RS or intrinsicoid deflection in leads from the right precordium and thought that the lateral wall of the right ventricle contributed little to the extrinsicoid deflection, or R wave, in these leads. They noted that, in some patients with right ventricular hypertrophy, a q wave preceded the late R wave in V_1 and that a q wave was absent in leads from the left chest, and suggested that in certain cases this was due to extreme rotation of the heart producing almost complete reversal of the electrical fields of the two ventricles so that the exploring electrode on the right chest had an indirect relationship to the surface of the left ventricle through the right atrium. However, they thought that in patients with mitral stenosis a large left atrium prevented such rotation. Sokolow and Lyon[4] also considered precordial Leads V_1 and V_5 to be of special value in the diagnosis of right ventricular hypertrophy, but mentioned that in the early stages of right ventricular hypertrophy in patients with mitral stenosis diagnostic electrocardiographic patterns were sometimes absent. In a later paper, Kossmann and associates[5] suggested that in patients with right ventricular hypertrophy, a late positive potential on the right side of the chest was due to forces derived from the crista supraventricularis. Goldberger[6] agreed that a tall R wave in the right precordial leads was a sign of right ventricular hypertrophy and also thought that a qR complex in Lead V_1 was produced

From The Regional Cardiovascular Centre, The City General Hospital, and the University Department of Medicine, The Royal Hospital, Sheffield, England.

Received for publication Jan. 23, 1956.

by extreme clockwise rotation of the heart. He believed that in these cases a unipolar lead on the anterior surface of the right chest faced the back of the heart and the epicardial surface of the left ventricle, and recorded a q wave from the left ventricular cavity and an R wave caused by the spread of the stimulus outward through the posterior wall of the left ventricle. Other views on the genesis of qR complexes in right chest leads from patients with right ventricular hypertrophy were discussed by Fowler and associates[7] who concluded, after studying intracavity electrocardiograms, that the delayed R wave of the qR complex was produced by activation of the free wall of the right ventricle.

The purpose of the present investigation was to study the ventricular patterns in right precordial leads from patients with mitral stenosis in order to assess their frequency and explain their genesis.

MATERIAL AND METHODS

Routine 12-lead electrocardiograms from 173 patients with mitral stenosis were examined and, in fifty-five of these, synchronous records from Leads V_1 and V_6 were obtained with an Elmqvist 3-channel instrument. The amplitude of the deflections was measured to the nearest 0.5 mm. Time intervals were measured to the nearest 0.01 second. In every case the presence of right ventricular hypertrophy was assessed clinically and radiologically.

RESULTS

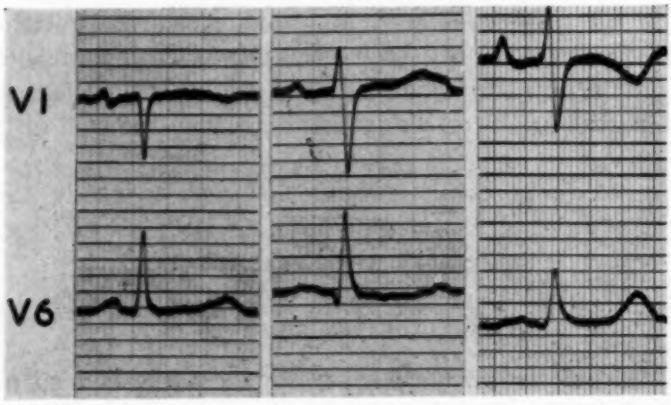
A wide range of electrocardiographic patterns were obtained; they are considered in the following groups:

1. In 104 cases there was a normal rS or S pattern in Lead V_1. The r wave was small and the r/S ratio less than 1.0. The simultaneous records obtained in this group of patients showed that the small r wave in V_1 was synchronous with the q wave in V_6 and its peak preceded that of the left ventricular R wave by about 0.02 second; the nadir of S in V_1 was synchronous with the peak of R or intrinsicoid deflection in V_6. In four patients the r wave was absent in Lead V_1 (Fig. 1). In sixty-seven patients there was clinical and radiologic evidence of right ventricular hypertrophy.

2. A single R wave or an Rs complex, with the R/s ratio greater than 1.0, occurred in twenty-one patients (Figs. 2 and 3). The R waves were from 2.5 to 24.0 mm. in height and the onset of the intrinsicoid deflection occurred at intervals of from 0.2 to 0.7 second. There was no correlation between the height of the R wave and the time of onset of the intrinsicoid deflection, which was delayed beyond 0.03 second in eight patients (Fig. 4). The duration of the entire complex did not exceed 0.11 second in any patient in this group. Five patients had auricular fibrillation and were receiving digitalis; all but one of the remaining sixteen patients showed RS-T segment depression and T-wave inversion in the V_1 lead. Synchronous records were done in five of the eight patients who showed delay in the intrinsicoid deflection in Lead V_1 and in all of these the peak of R in V_1 occurred later than the peak of R in V_6 by intervals

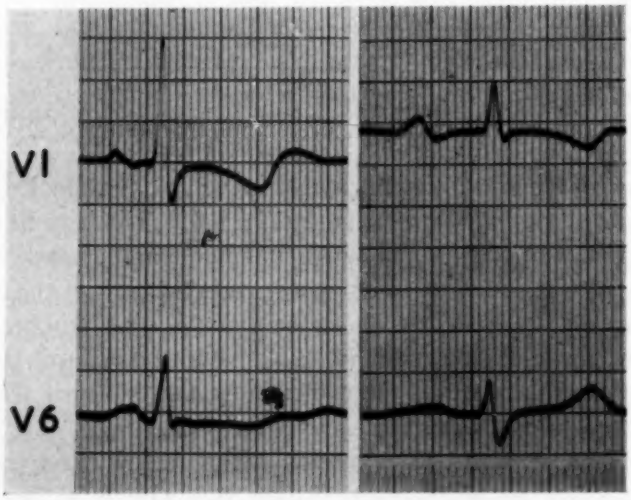
up to 0.02 second (Fig. 3, *B* and *C*). Right ventricular hypertrophy was diagnosed clinically in all the patients in this group and was confirmed on radiologic examination in fifteen.

3. In thirty-six cases there was an RSR′ pattern in V_1 (Figs. 5 and 6,*A*).



A. *B.* *C.*

Fig. 1.—*A* to *C*, Synchronous records of Leads V_1 and V_6 from three patients with mitral stenosis showing normal S and rS patterns in Lead V_1.



A. *B.*

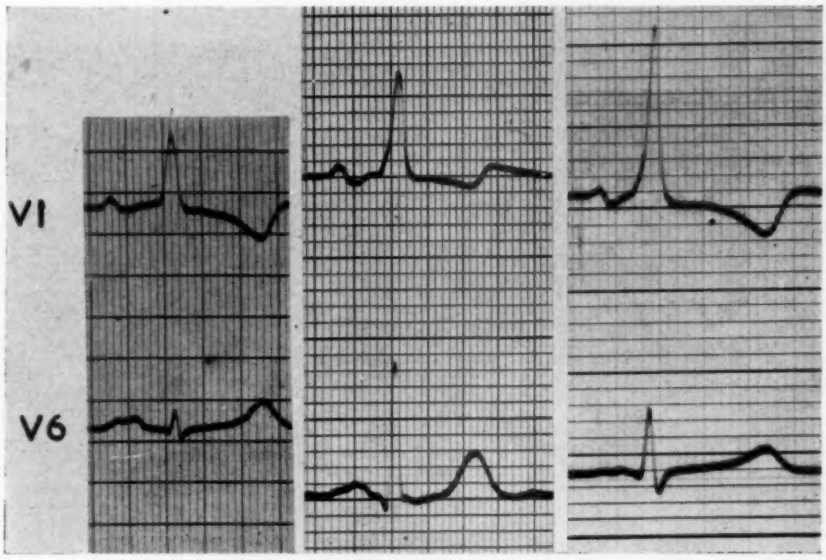
Fig. 2.—*A* and *B*, Leads V_1 and V_6 recorded synchronously from two patients with mitral stenosis showing Rs complexes in V_1. In *A* the intrinsicoid deflection in V_1 occurs earlier than in V_6. In *B* the intrinsicoid deflection in V_1 occurs later than in V_6.

The form of these complexes differed greatly owing to variations in height of the primary and secondary R waves, and the depth of the S wave, but it was possible to define three main groups:

A. In some, a small secondary r′ wave appeared as a notch on either limb of the S wave and its peak was below the isoelectric line. This pattern was present in eleven cases. The height of the primary R wave varied from less

than 1.0 mm. to 5.0 mm. The intrinsicoid deflection, measured from the onset of the upstroke of R to the peak of R′ was delayed beyond 0.03 second in all, and had a maximum of 0.08 second, but there was no prolongation of QRS.

B. In some the secondary R wave occurred as a positive deflection, and



A. *B.* *C.*

Fig. 3.—*A* to *C*, Synchronous records of Leads V_1 and V_6 in three patients with mitral stenosis showing tall R waves in V_1. In *A* the intrinsicoid deflection in V_1 = 0.03 second and occurs a brief interval earlier than in V_6. In *B* and *C* the V_1 leads show delay in the intrinsicoid deflections. In *C* the peak of R in V_1 is synchronous with the nadir of S in V_6.

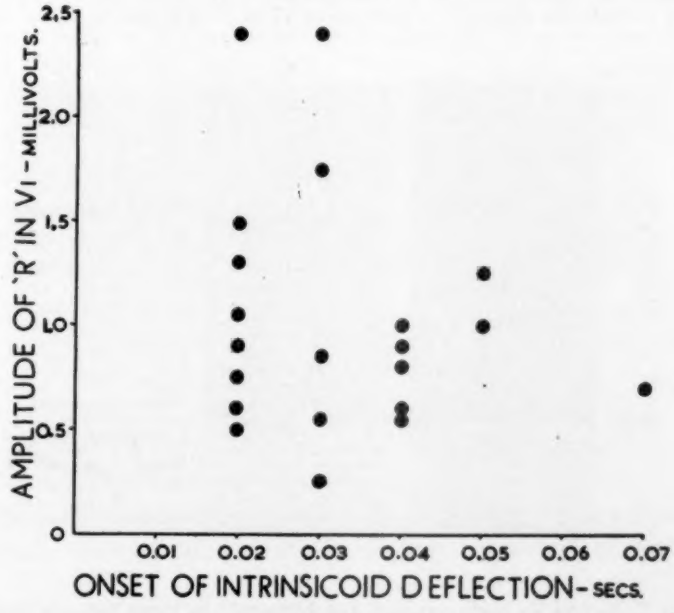
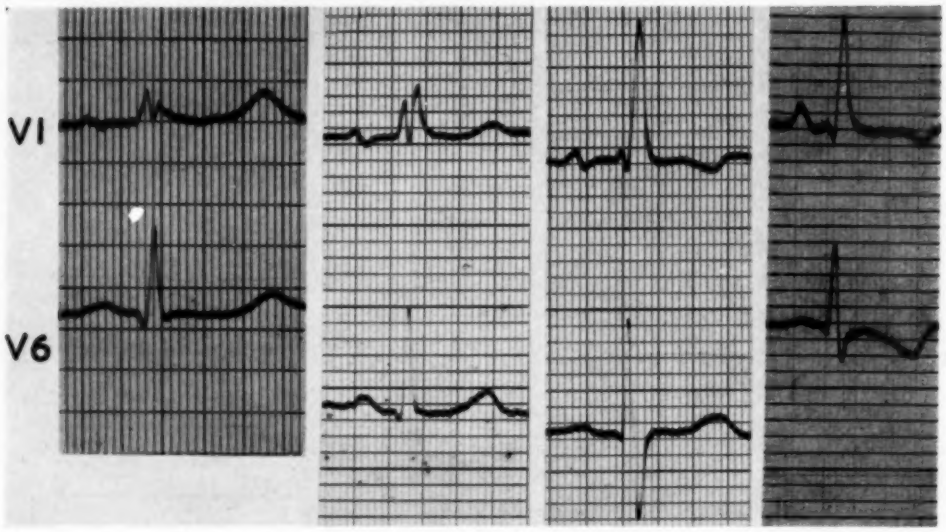


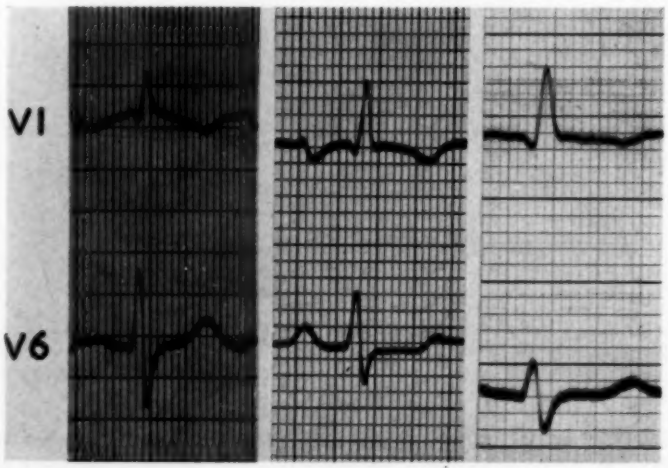
Fig. 4.—Scatter diagram of amplitude of R against time of onset of intrinsicoid deflection in Rs and R complexes in Lead V_1 in twenty-one patients with mitral stenosis.

extended above the isoelectric line but was smaller than the primary R wave (Fig. 5,*A*). This pattern occurred in eight patients. The R waves were from 1.0 to 12 mm. in height and the R′ waves from less than 1.0 to 9.5 mm. The intrinsicoid deflection was delayed beyond 0.03 second in all patients and in none was there any prolongation of QRS.



A. *B.* *C.* *D.*

Fig. 5.—*A* to *D*, V_1 and V_6 recorded synchronously in four patients with mitral stenosis showing various RSR′ complexes in the V_1 leads. There is relative increase in height of the secondary R′ wave and decrease in height of the primary R wave in *A* to *D*. In all the tracings the onset of the secondary R′ wave in V_1 occurs at or near the intrinsicoid deflection in V_6. In *C* and *D* the small primary R waves in V_1 are synchronous with q waves in V_6 and the tall secondary R′ waves are associated with deep S waves in V_6. The intrinsicoid deflection is delayed in V_1 in *A* to *D* and in *C* the duration of QRS in V_1 = 0.12 second.



A. *B.* *C.*

Fig. 6.—*A* to *C*, V_1 and V_6 leads recorded synchronously in three patients with mitral stenosis. *A* shows an RSR′ complex with a very small primary r wave and a tall secondary R′ wave in V_1, and a deep S wave in V_6. *B* and *C* show qR complexes in V_1 and complexes with deep S waves in V_6. The S wave of the V_1 RSR′ complex in *A* and the V_1 q waves in *B* and *C* have the same time relationship to the peak of R in V_6.

C. In others R′ was taller than R, and complexes of this type occurred in seventeen patients (Fig. 5, *B*, *C*, and *D*, and Fig. 6, *A*). The secondary R′ waves were from 1.5 to 15.5 mm. in height. The primary R waves were small and did not exceed 4.0 mm., being less than 1.0 mm. in height in eight cases. Delay in the intrinsicoid deflection was present in all and in two patients QRS was prolonged to 0.12 second, indicating right bundle branch block (Fig. 5,*C*); in these R′ was 6.5 and 16.5 mm. and R was, in each case, less than 2.0 mm.

It was noted that the tall secondary R′ waves occurring in group C above were usually associated with deep S waves in V_6, where the complexes had the appearance of those usually found in the transition zone (Figs. 5,*C* and *D*, and 6,*A*). In these patients the simultaneous leads showed the following features:

A. When a q wave was present in V_6, its onset occurred synchronously with the onset of the primary R in V_1 (Fig. 5,*A* to *D*).

B. Where the primary R in V_1 was less in height than 2.0 mm. its peak was synchronous with the nadir of q in V_6 (Fig. 5,*C* and *D*).

C. When the magnitude of the primary R in V_1 was greater than that of q in V_6, its peak occurred later than the nadir of q, but the interval was usually less than 0.02 second (Fig. 5,*A* and *B*).

D. The nadir of S in V_1 was synchronous with the peak of R in V_6 (Fig. 5,*A* to *D*).

E. Where R′ was taller than R in V_1, its peak was synchronous with the nadir of S in V_6 (Fig. 5,*C* and *D*).

There was clinical and radiologic evidence of right ventricular hypertrophy in all thirty-six patients in this group with RSR′ patterns in Lead V_1.

4. In nine patients there was a qR complex in Lead V_1 (Fig. 6,*B* and *C*). In all, the intrinsicoid deflection was delayed beyond the normal 0.03 second, and varied from 0.04 to 0.06 second, and the duration of the complexes was prolonged to 0.12 second in two cases. The form of the complexes in Lead V_6 varied; in three patients qRs patterns were obtained and in two cases deep, wide S waves were recorded (Fig. 6,*C*). The synchronously recorded leads showed that the q and R waves in Lead V_1 had the same time relation to the components of the V_6 complexes as the S and R′ waves in patients showing RSR′ complexes in Lead V_1 (Fig. 6,*A* to *C*). The q wave in Lead V_1 was synchronous with the peak of R in Lead V_6, and R in Lead V_1 was synchronous with the nadir of S in Lead V_6 (Fig. 6,*B* and *C*). When present, the q wave in V_6 preceded the q in V_1 by about 0.02 second. In five patients in this group RSR′ complexes were recorded in either Lead V_{3R} or V_2 and in these the SR′ component of the RSR′ complex was coincident with the qR pattern in Lead V_1.

Evidence of right ventricular hypertrophy was present on clinical and radiologic examination of all the patients in this group.

5. In three cases polyphasic, RSRSRS patterns were present in Lead V_1. All three patients showed clinical and radiologic evidence of right ventricular hypertrophy.

DISCUSSION

It is now generally believed that an rS complex in Lead V_1 in normal subjects is due to septal activation, which produces the r wave, and to the resultant of

the potentials developed during activation of the ventricles, which produces a deep S wave, since the left ventricle is dominant. However, while Kossmann and associates[5] accepted that the initial part of the r wave in Leads V_1 and V_2 was due to septal forces, they thought that activation of the free wall of the right ventricle produced the final part of the primary r wave. Goldberger[6] and Kossmann[5] found that there was occasionally a final r′ in Lead V_1 in normal subjects, which they thought was due to late stimulation of part of the right ventricle, and Rosenman[8] noted this in about 5 per cent of normal records. When other right precordial leads have also been studied a final r′ wave has frequently been recorded; Mounsey and associates[9] noted secondary r′ waves of "embryonic" form in Leads V_1 and V_{4R} in fourteen of thirty healthy men, and Camerini and Davies[10] found complexes with secondary r′ waves in one or more of the right chest leads in forty-two of fifty normal subjects.

In the present series 104 patients had rS or S complexes in Lead V_1, although sixty-seven of these had clinical and radiologic evidence of right ventricular hypertrophy. Kossmann and associates[3] also found normal right precordial electrocardiograms in two patients with mitral stenosis with good clinical evidence of right ventricular hypertrophy, and Fowler and associates[7] noted that V leads from the right precordium sometimes showed normal rS patterns in patients with right ventricular hypertrophy. Goldberger[6] thought that the increased mass of the right ventricle in right ventricular hypertrophy tended to counterbalance the electrical activity of the left ventricle, but he believed that rS patterns occurred in precordial leads facing the epicardial surface of the hypertrophied right ventricle when the stimulus passed through the main muscle mass at right angles away from the recording electrode, and also when there was marked clockwise rotation of the heart. Rosenman[8] pointed out that so long as there is left ventricular dominance, as in normal subjects, the forces of right ventricular activation are not represented as positive deflections but only lessen the amplitude of S waves in the right chest leads and R waves in the left chest leads, and it seems probable that the increase in right ventricular potentials associated with right ventricular hypertrophy are often obscured by normal left ventricular forces. When there is associated left ventricular hypertrophy, Pagnoni and Goodwin[11] noted that the electrocardiographic signs of right ventricular hypertrophy were sometimes masked and it is noteworthy that both patients with mitral stenosis reported by Kossmann and associates[3] and referred to above, also had mitral incompetence and one had aortic incompetence. In the present series it seems probable that electrical changes due to right ventricular hypertrophy were masked in many cases since these occurred synchronously with left ventricular excitation. Where there was also mitral or aortic incompetence, left ventricular hypertrophy caused by these lesions would further secure the masking effect of left ventricular potentials. Wilson and associates[1] thought that hypertrophy of the right ventricle might abolish or reverse the normal difference between it and the left and increase the height of the r wave over the right chest. Although right ventricular hypertrophy would have to be of considerable magnitude for the potentials in its free wall to outweigh the normal left ventricular preponderance, it would be difficult, using the criteria

at present accepted, to assess this hypertrophy from changes produced in the height of the r wave. Myers and associates[12] recorded r waves from 1.0 to 7.0 mm. in height in V_1 in their series of normal subjects, and Sokolow and Lyon[4] also regarded 7.0 mm. as the maximum normal height of the r wave in V_1. The criteria of the latter authors[4] for the diagnosis of right ventricular hypertrophy, which were based on the records of sixty patients of whom forty-four had right ventricular hypertrophy due to congenital heart disease, are probably not applicable to patients with mitral stenosis. Congenitally malformed hearts frequently show right ventricular hypertrophy greatly in excess of that encountered in acquired heart disease such as mitral stenosis and it therefore appears that in the latter right ventricular hypertrophy may produce electrocardiographic changes less striking than those regarded as diagnostic by Sokolow and Lyon.[4] There was clinical evidence of right ventricular hypertrophy in all twenty-one patients and radiologic evidence of right ventricular hypertrophy in fifteen patients in this series who showed Rs or R complexes in V_1, although the electrocardiographic criteria for the diagnosis of right ventricular hypertrophy were not completely fulfilled in any. These findings suggest that further studies correlating electrocardiographic appearances with post-mortem evidence of right ventricular hypertrophy are necessary to establish some criteria for the electrocardiographic diagnosis of right ventricular hypertrophy in patients with mitral stenosis. In this group the S wave in V_1 was absent or small and in some patients the peak of the R wave was delayed. The synchronous leads showed that in five patients the peak of the R in V_1 occurred later than the peak of the R in V_6 (Figs. 2*B*, and 3,*B* and *C*). However, such a delay in the R wave should not be regarded as an invariable feature associated with right ventricular hypertrophy. Rosenman[8] mentioned that in some patients with right ventricular hypertrophy the peak of R in V_1 still occurred earlier than in V_5 or V_6, and Goldberger[6] stated that the time of onset of the intrinsicoid deflection may be normal in Lead V_1 when there is hypertrophy of the right ventricle. As already mentioned, the time of onset of the intrinsicoid deflection was not directly related to the height of the R wave in the present group (Fig. 4). Furthermore, high values for the R/s ratio were sometimes associated with R waves of less than 7.0 mm. in height. The relative importance of the voltage of R and the time of onset of the intrinsicoid deflection in the electrocardiographic diagnosis of right ventricular hypertrophy is difficult to assess. The rapid upstroke to a height of 7.0 mm. or more, of an R wave which reaches its peak within 0.03 second and therefore exceeds simultaneous left ventricular forces, may represent a greater degree of right ventricular hypertrophy than an R wave of the same magnitude, part or all of which is written when activation of the left ventricle is complete.

Among the thirty-six patients in the present series showing RSR′ complexes in V_1, there were twenty in whom the patterns resembled those obtained over the right precordium in normal subjects by Camerini and Davies.[10] In many of these twenty patients the S wave was the most prominent deflection and in none did the primary R wave exceed 7.0 mm. in height and R′ was either of "embryonic" form, appearing as a notch on the S wave, or was of small voltage.

The duration of QRS did not exceed 0.11 second in any. Clinical and radiologic examination provided evidence of right ventricular hypertrophy in all of these twenty patients, but in view of the frequent occurrence of this type of RSR′ pattern in normal subjects it cannot be considered an electrocardiographic sign of right ventricular hypertrophy. Mounsey and associates[9] thought it possible that the "embryonic" r wave in normal hearts might, with the development of right ventricular hypertrophy, sometimes increase in voltage until it became the secondary R′ wave of the RSR′ pattern. Camerini and Davies[10] suggested that the secondary R′ waves recorded from right chest leads in normal subjects are due to activation of the conus of the right ventricle. Small rs deflections were followed by R′ waves of height greater than 7.0 mm. in five of the present patients, all of whom had clinical and radiologic signs of right ventricular hypertrophy, and it is possible that the tall R′ waves occurring in these patients with mitral stenosis are due to potentials developed in a hypertrophied conus. Kossmann and associates,[5] however, believed that in normal subjects the secondary R′ wave recorded from the right chest is due to stimulation of the crista supraventricularis. They suspected that hypertrophy of this part occurred in conditions causing increased work of the right ventricle and was responsible for some of the late positive deflections found in right precordial leads in right ventricular hypertrophy. Myers and associates[2] thought that the R′ of the RSR′ complex was due to activation of the outer wall of the right ventricle and regarded such complexes as indicating incomplete right bundle branch block when the duration of QRS was less than 0.10 second. Mounsey and associates,[9] however, stated that there was no good evidence that incomplete right bundle branch block contributed to the development of RSR′ since no essential difference in timing was noted between the secondary R′ waves and the embryonic r waves from which they appeared to grow. Goldberger[6] mentioned the appearance of a small rs deflection preceding the main tall upward deflection and considered that the final R′ was due to the spread of the stimulus outward through the wall of the hypertrophied right ventricle. When right bundle branch block is present, the tall secondary R′ wave is explained by the slow passage of the stimulus outward through the wall of the right ventricle at a time when stimulation of the left ventricle is practically complete.[6] In the present series, synchronous records show that the onset of the secondary R′ wave occurs almost simultaneously with the onset of the intrinsicoid deflection in V_6 whether or not right bundle branch block is present (Fig. 5), and it seems unnecessary to postulate a separate genesis for the tall secondary R′ wave in a complex of briefer duration than the arbitrary limit of 0.12 second required for the diagnosis of "block." Examination of hearts at autopsy shows that gross thickening of the trabeculae within a relatively thin layer of myocardium is responsible for the hypertrophy of the right ventricular wall in many patients with mitral stenosis. This state of affairs is in striking contrast to the thick wall of solid myocardial tissue which is seen in right ventricular hypertrophy when it occurs in congenital heart disease. It is possible that the tall, late R waves in Lead V_1 in patients with mitral stenosis may be due to the delayed spread of the stimulus through a right ventricular wall which is largely composed of hypertrophied trabeculae. Certainly the

anatomic preponderance of the right ventricle characteristic of some forms of congenital heart disease and associated with tall primary R waves and little or no delay in the intrinsicoid deflection is rarely seen in mitral stenosis.

The synchronous records in the present series show that the qR and RSR′ complexes obtained from Lead V_1 are essentially the same. In qR and RSR′ patterns the R and R′ waves, respectively, commence at or very near the onset of the intrinsicoid deflection in V_6 (Fig. 6). The initial R of RSR′ is in some cases a very small upward deflection (Fig. 6,*A*) and Myers[13] suggested that the qR patterns resulted when the r wave was lost in transmission to the precordium or was absent due to initial negativity of the right ventricular cavity. Mounsey and associates[9] also thought that the disappearance of an initial R wave in right chest leads, which occurred concurrently with the growth of an embryonic r wave, changed the final picture from an RSR′ pattern to a qR pattern of right ventricular hypertrophy. In the absence of the R wave, the initial deflection is negative and is termed a q wave, although it is manifestly different from a q wave recorded in Lead V_6. The q of qR in Lead V_6 occurs earlier and is the result of septal activation, whereas the q in V_1, which has the same time relation to the records in V_6 as the S of the RSR′ patterns in V_1, is the result of the left ventricular forces responsible for the R wave in V_6. The late R wave of the V_1 qR complex has the same genesis as the R′ wave of the RSR′ complex; namely, that it is produced by growth of an "embryonic" or small r wave found frequently in normal subjects when the free wall of the right ventricle undergoes hypertrophy. There is no support for the view that a qR complex recorded from Lead V_1 in these patients is derived from potentials developed in the cavity and wall of the left ventricle which, by rotation, comes to face the right precordial lead.

SUMMARY

1. Routine 12-lead electrocardiograms were recorded in 173 patients with mitral stenosis, 136 of whom had right ventricular hypertrophy assessed clinically and radiologically. In fifty-five of these patients Leads V_1 and V_6 were recorded synchronously.

2. A great variety of QRS patterns were obtained from precordial Lead V_1 and these fell into five groups:

A. Clinical and radiologic evidence of right ventricular hypertrophy was present in sixty-seven of the 104 patients who showed normal rS or S complexes in Lead V_1.

B. Rs complexes or single R waves occurred in Lead V_1 in twenty-one patients, all of whom had clinical evidence of right ventricular hypertrophy which was confirmed radiologically in fifteen cases. There was no relationship between the height of the R wave and the time of onset of the intrinsicoid deflection in these twenty-one cases, and the criteria for the electrocardiographic diagnosis of right ventricular hypertrophy were not completely fulfilled in any.

C. RSR′ patterns were obtained in thirty-six patients, all of whom had clinical and radiologic evidence of right ventricular hypertrophy, but in twenty the form of the complexes was similar to that sometimes obtained in normal

subjects. In all patients there was delay in the intrinsicoid deflection and in two right bundle branch block was present.

D. A qR complex was recorded from V_1 in nine patients, all of whom showed clinical and radiologic evidence of right ventricular hypertrophy. Delay in the onset of the intrinsicoid deflection occurred in all.

E. Polyphasic complexes occurred in three patients who showed right ventricular hypertrophy on clinical and radiologic examination.

3. It is probable that in many patients in this series, the degree of right ventricular hypertrophy was insufficient to produce forces capable of outweighing those incident to simultaneous left ventricular excitation. The absence of classical electrocardiographic signs of right ventricular hypertrophy in any patient in Groups 1 and 2 of the present series suggests that there is a need for the clear definition of the electrocardiographic signs of right ventricular hypertrophy in mitral stenosis.

4. The lack of correlation between the height of the R wave and the time of onset of the intrinsicoid deflection in the V_1 Rs and R complexes suggests that delay in onset of the intrinsicoid deflection is not a reliable sign of right ventricular hypertrophy in mitral stenosis.

5. Synchronous leads showed an essential similarity between RSR′ and qR complexes in Lead V_1. Evidence is presented to show that a qR pattern in right precordial leads in patients with mitral stenosis is not due to potentials derived from the left ventricle.

6. The tall secondary R′ waves of RSR′ complexes and the R waves of qR complexes were considered to be due to the slow passage of the stimulus through the wall of the right ventricle when stimulation of the left ventricle was almost complete. It is suggested that in mitral stenosis the slow passage of the stimulus is related to the type of thickening of the right ventricular wall, which in this disease consists largely of hypertrophied trabeculae.

Our thanks are due to Dr. James W. Brown, Mr. J. T. Chesterman, and Professor Stuart-Harris for facilities in their departments and to Miss Nancy Hardy for valuable technical assistance.

REFERENCES

1. Wilson, F. N., Johnston, F. D., Rosenbaum, F. F., Erlanger, H., Kossmann, C. E., Hecht, H., Cotrim, N., de Oliveira, R. M., Scarsi, R., and Barker, P. S.: Am. Heart J. **27**:19, 1944.
2. Myers, G. B., Klein, H. A., and Stofer, B. E.: Am. Heart J. **35**:1, 1948.
3. Kossmann, C. E., Berger, A. R., Brumlik, J., and Briller, S. A.: Am. Heart J. **35**:309, 1948.
4. Sokolow, M., and Lyon, T. P.: Am. Heart J. **38**:273, 1949.
5. Kossmann, C. E., Berger, A. R., Rader, B., Brumlik, J., Briller, S. A., and Donnelly, J. H.: Circulation **2**:10, 1950.
6. Goldberger, E.: Unipolar Lead Electrocardiography and Vectorcardiography, London, 1953, Henry Kimpton.
7. Fowler, N. O., Westcott, R. N., and Scott, R. C.: Circulation **5**:441, 1952.
8. Rosenman, R. H.: Am. Heart J. **40**:522, 1950.
9. Mounsey, J. P. D., Ritzmann, L. W., and Selverstone, N. J.: Brit. Heart J. **14**:442, 1952.
10. Camerini, F., and Davies, L. G.: Brit. Heart J. **17**:28, 1955.
11. Pagnoni, A., and Goodwin, J. F.: Brit. Heart J. **14**:451, 1952.
12. Myers, G. B., Klein, H. A., Stofer, B. E., and Hiratzka, T.: Am. Heart J. **34**:785, 1947.
13. Myers, G. B.: Circulation **1**:860, 1950.

THE DIAGNOSTIC SIGNIFICANCE OF TALL UPRIGHT T WAVES IN THE CHEST LEADS

J. FREUNDLICH, M.D.

VANCOUVER, BRITISH COLUMBIA

LARGE upright T waves in the limb and chest leads have occasionally been reported and considered as a sign of a myocardial lesion.[1,2] Wood and Wolferth[3] described huge T waves in the chest leads as an important diagnostic feature of an acute or subacute cardiac infarction or as a sign of a healing anterior infarction. However, in the majority of the reported cases, the high T waves were not the only abnormal sign in the electrocardiogram; they also have been associated with the changes in the QRS or in S-T segments. Only recently Dressler and Roesler[4] have renewed the interest in the importance of high T waves as the outstanding sign in the earliest stage of myocardial infarction.

The present paper is a report of 110 cases in which very high T waves, especially in the chest leads, have been the only abnormal sign in the electrocardiogram. While in seventeen cases these high T waves were the forerunners of the characteristic electrocardiographic changes in myocardial infarction, the majority of the cases presented here displayed high T waves in the chest leads, which persisted for months or years and have been obtained from patients who had frequent anginal attacks. The study to be reported was undertaken to determine the diagnostic value of the large T waves on the basis of clinical and post-mortem findings.

CASE REPORTS

CASE 1.—C. T., a 50-year-old man, was well until Feb. 12, 1947. That morning as he was walking he was seized with a sudden agonizing pain across the chest, radiating to both arms. On admission to the hospital about one-half hour later the patient was very pale and perspired profusely. The blood pressure was 120/90 mm. Hg, the heart sounds normal, and sedimentation rate and white blood count were normal.

The first electrocardiogram (Fig. 1,*A*) obtained about one-half hour after the onset of the attack showed abnormally high T waves in standard and in the chest leads, otherwise no abnormal signs. Electrocardiograms repeated on this day remained essentially unchanged.

On the following day the electrocardiogram (Fig. 1,*B*) showed an elevation of RS-T in the standard and in the chest leads and marked change of QRS in Leads II and III. Four days later the T waves became sharply inverted in the limb and chest leads (Fig. 1,*C*).

Summary: A 50-year-old man suffered a severe attack of precordial pain. The electrocardiogram taken during the attack showed strikingly high T waves in the standard and in the chest leads. Twenty-four hours later the electrocardiogram revealed signs of a recent combined anterior and posterior wall infarction.

From the Department of Medicine, St. Paul's Hospital, Vancouver, B.C.
Received for publication Jan. 25, 1956.

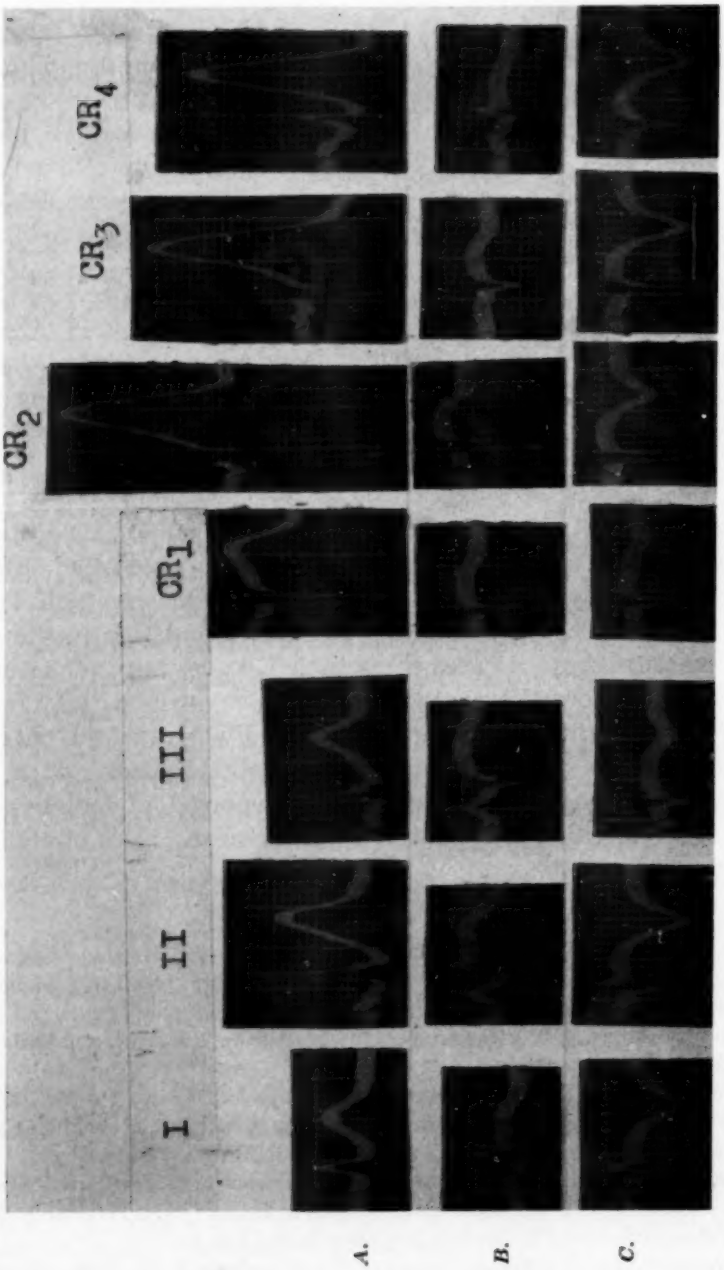


Fig. 1.—Case 1. *A* (Feb. 12, 1947), obtained half an hour after the onset of symptoms. Huge T waves in standard and chest leads (amplitude 20 mm.) without abnormal elevation of S-T. *B* (Feb. 13, 1947), elevation of RS-T in standard and chest leads, marked change of QRS in Leads II and III, decrease of the amplitude of T. *C* (Feb. 17, 1947), inversion of T in standard and chest leads. Combined anterior and posterior wall infarction.

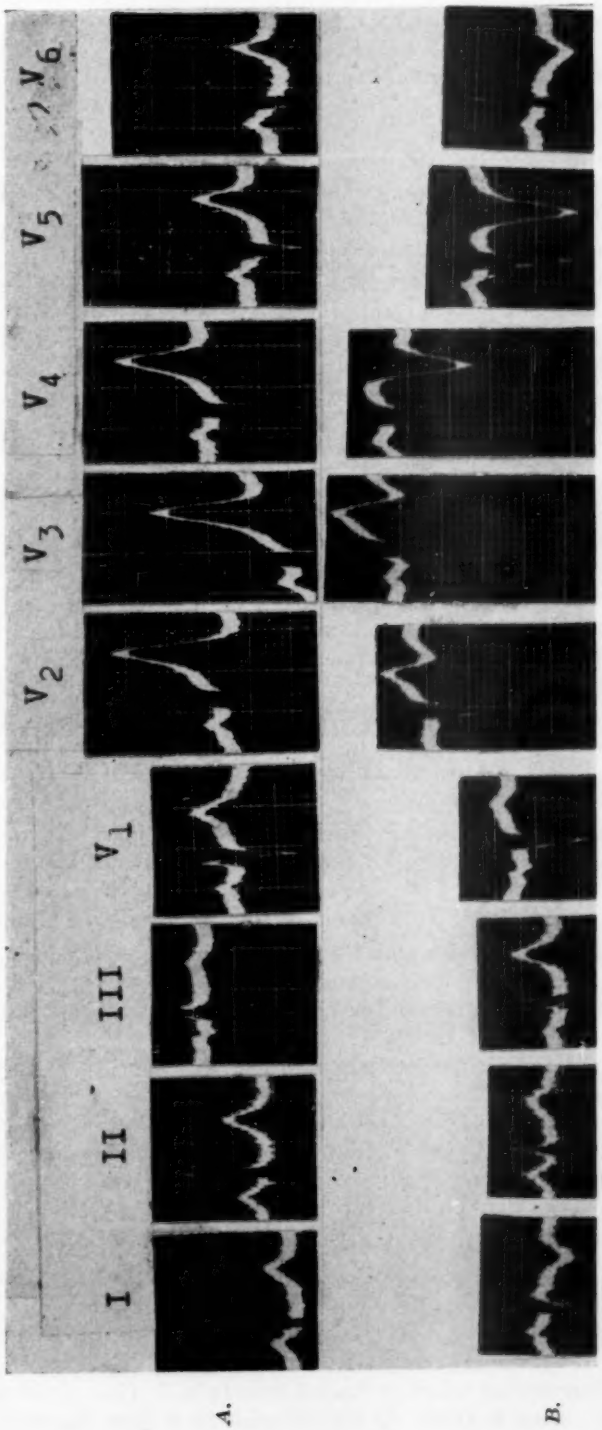


Fig. 2.—Case 2. *A* (March 31, 1952), shows high T waves in V_2 (amplitude 14 mm.), V_3 (14 mm.), V_4 (11 mm.) without changes of QRS. *B* (Jan. 15, 1954), inverted T in Lead I and in the chest leads, QS complex in chest leads; signs diagnostic of anteroseptal infarction.

CASE 2.—W. C., a 49-year-old man, was first seen on March 31, 1952. He gave a two-year history of precordial pain on exertion. The physical and fluoroscopic findings were normal, blood pressure 150/70 mm. Hg. The electrocardiogram (Fig. 2,*A*) showed high T waves in the chest leads (V_2, V_3, and V_4) as the only outstanding abnormality. During the following year (1953) the electrocardiogram remained essentially unchanged. On Jan. 2, 1954, while at work the patient suffered an intense precordial pain. He felt very weak afterward and remained at home for four weeks. He was seen about four weeks after the attack. The physical and fluoroscopic findings were unchanged as compared with those in 1952, blood pressure 130/80 mm. Hg, and sedimentation rate 36 mm. per hour. The electrocardiogram (Fig. 2,*B*) showed inverted T in Lead I and in the chest leads and QS complex in V_{1-5}.

Summary: A 49-year-old man had suffered frequent anginal attacks on exertion during a two-year period. The electrocardiograms taken repeatedly during this period displayed high T waves in the chest leads. After the last attack the high T waves became inverted and the signs of anteroseptal infarction became evident.

CASE 3.—J. M. H., a 61-year-old man, was in good health until 1950 when he noticed a pressure across the chest while chopping wood. Since then he has had frequent precordial pain on exertion. The patient was first seen on Jan. 5, 1952. The physical and fluoroscopic findings were normal, blood pressure 150/60 mm. Hg.

The electrocardiogram (Fig. 3,*A*) showed high T in V_2 and V_3, otherwise no abnormal signs. This ECG pattern remained unchanged until Aug. 1, 1952, when the patient suffered a severe anginal attack. He was admitted to hospital about four hours later and on admission the physical and x-ray findings were normal, the blood pressure was 150/60 mm. Hg, while blood count normal, sedimentation rate not increased, Kahn negative.

The electrocardiogram (Fig. 3, *B*) obtained about four hours after the attack showed inverted T in V_2, V_4, and V_5, while R remained unchanged in the chest leads, suggesting a septal infarction.

During the following two years the patient was relatively well. On Sept. 18, 1954, he suffered a severe precordial pain and on admission to hospital the electrocardiogram revealed an acute posterior infarction (Fig. 3, *C*). The patient died two days later and the autopsy demonstrated a large recent hemorrhagic infarction of the posterior wall of the right ventricle and many pale areas of the anterior septal portion, indicating old scarring due to previous myocardial infarction.

Summary: Very high T waves in the chest leads were the only abnormal sign in electrocardiogram of a patient who had suffered from anginal attacks. The high T waves subsequently became inverted, indicating septal infarction. Following another severe anginal attack the electrocardiogram showed an acute posterior infarction. The post-mortem examination confirmed the diagnosis.

CASE 4.—B. B. was a 69-year-old man. He was well until June 21, 1954. That morning, while driving a car, he suffered a severe pain in the chest radiating down the left arm. The patient was seen four hours later. He was pale and perspired profusely, the blood pressure was 80/50 mm. Hg, the second aortic sound faint.

The electrocardiogram taken immediately showed high T waves in V_2 and V_3, otherwise no abnormal signs (Fig. 4, *A*). The second electrocardiogram was taken on the following day, twenty-four hours later. It shows (Fig. 4, *B*) small Q and inverted T in Lead I, marked Q waves in V_4, V_5, and V_6, and inverted T in V_5 and V_6. The amplitude of T in V_2 and V_3 decreased.

Summary: The electrocardiogram obtained four hours after a severe anginal attack showed high T in the chest leads as the only abnormal sign. Twenty-four hours later the second tracing displayed the characteristic signs of anterolateral infarction.

CASE 5.—S. O. L., a 56-year-old man, was first seen on April 5, 1946. He was in good health until 1945 when he began to have chest pain on exertion extending to the left arm. On examination the heart was not enlarged, blood pressure 140/80 mm. Hg, sedimentation rate and white cell count normal.

The first electrocardiogram was taken on April 5, 1946 (Fig. 5, *A*). It shows a very high T in CR_3 and CR_4, and no change in QRS. In the following three years electrocardiograms were repeatedly taken and did not show any essential change in the tracings.

On Jan. 23, 1949, the patient suffered an attack of severe retrosternal pain. He was admitted to the hospital a week later. On admission the patient had a slight dyspnea, blood pressure was 118/80 mm. Hg, white cell count was normal, and sedimentation rate not increased.

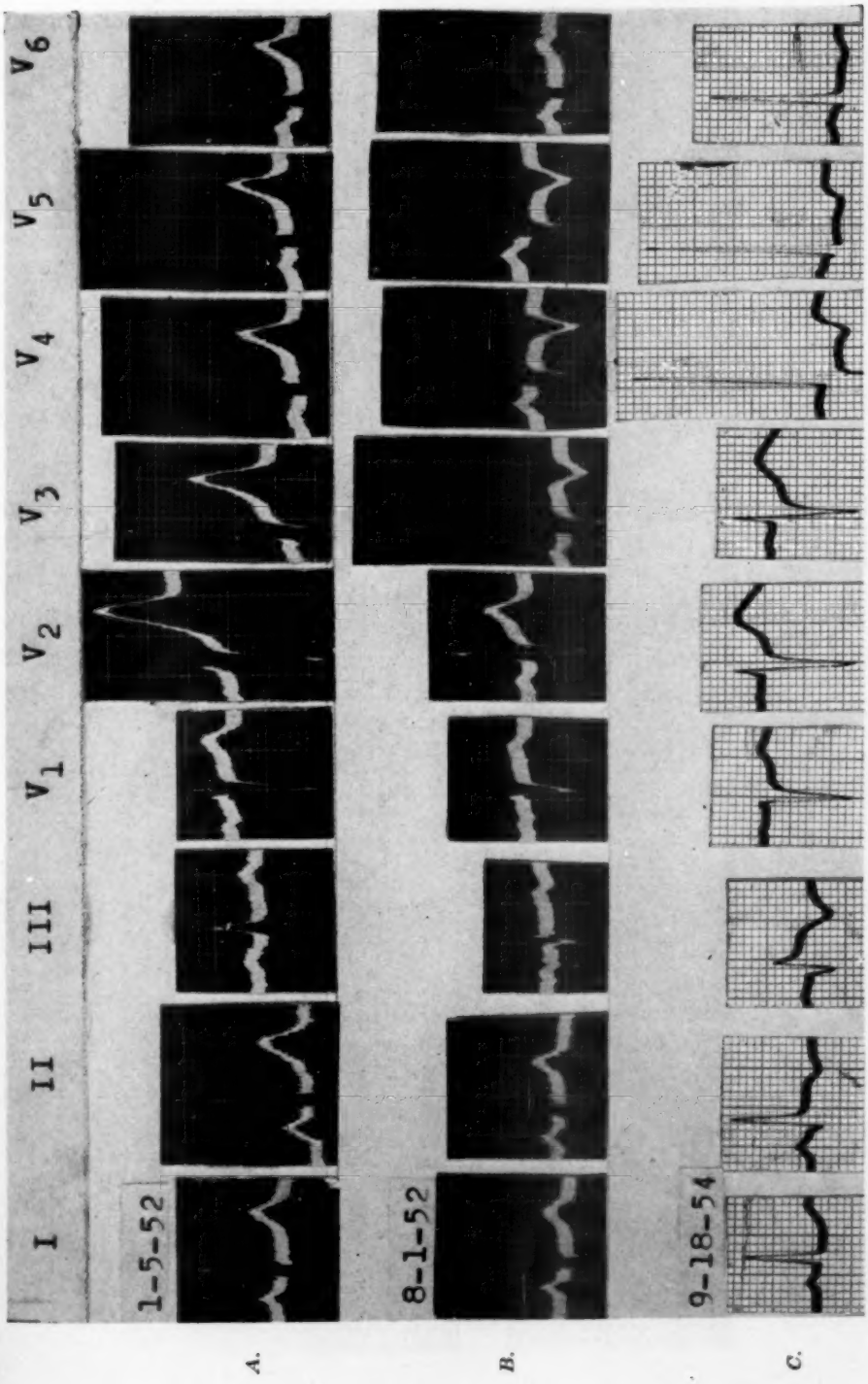


Fig. 3.—Case 3. *A* shows high T waves in V_2 (amplitude 11 mm.) and in V_3 (11 mm.), no changes in QRS. *B*, taken four hours after the attack, shows inversion of the high T waves in V_2, V_4, and V_5, while R in the chest leads persists. Signs suggestive of septal infarction. *C*, acute posterior wall infarction.

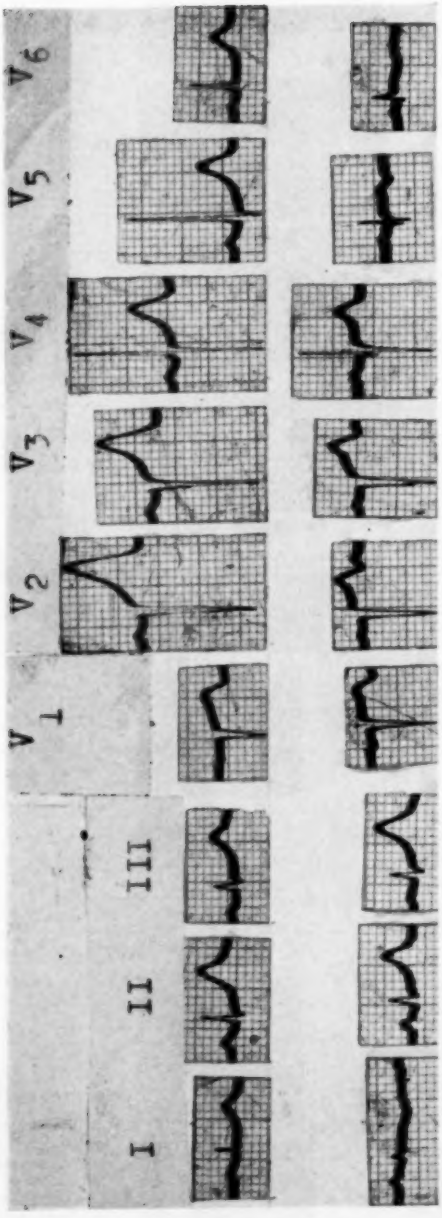


Fig. 4.—Case 4. *A* (June 21), taken four hours after the onset of symptoms. High T waves in V_2 and V_3. *B* (June 22), twenty-four hours later. Small QRS and inverted T in Lead I, marked Q in V_4, V_5, and V_6, inverted T in V_5 and V_6. Signs pointing to anterolateral infarction.

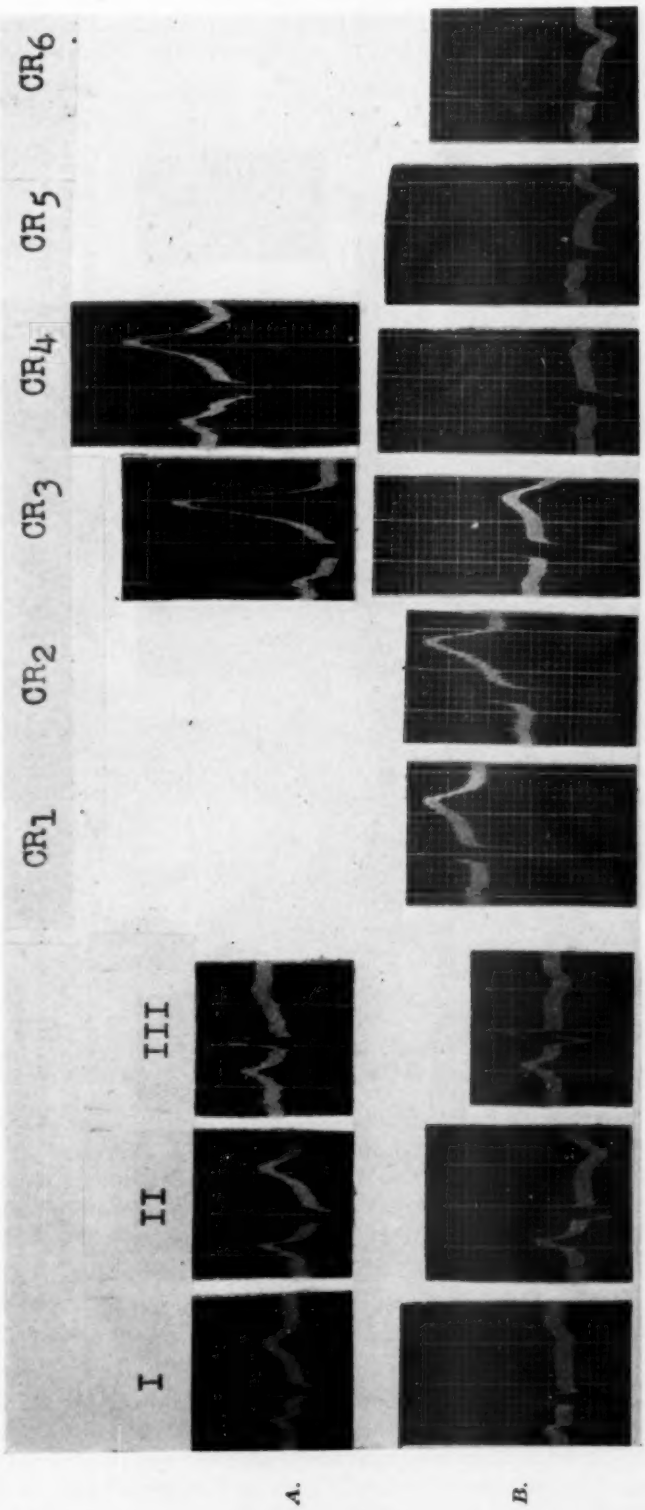


Fig. 5.—Case 5. *A* (April 5, 1946), very high T waves in CR_3 (amplitude 20 mm.) and CR_4 (amplitude 13 mm.). *B* (Jan. 23, 1949), signs of posterior wall infarction, decrease of the amplitude of the T waves.

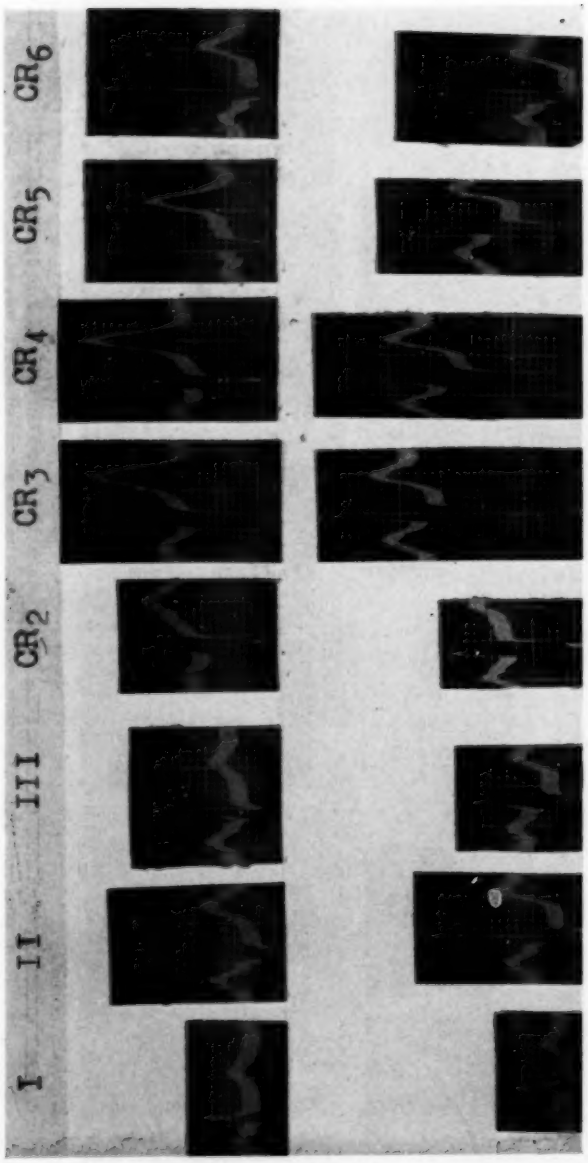


Fig. 6.—Case 6. *A* (May 5, 1948), tall peaked T waves in CR_3 (amplitude 12 mm.), CR_4 (amplitude 13 mm.), CR_5 (amplitude 10 mm.), no change in QRS. *B* (May 13, 1948), taken one hour after an attack, shows a marked depression of RS-T in standard and chest leads (no digitalis was given), and decrease of the amplitude of the T waves. Tracing suggestive of subendocardial lesion.

The electrocardiogram, obtained eight days after the attack, showed a deep Q in Leads II and III, inverted T in Lead I, sharply inverted T in II and III, decrease in the amplitude of the T waves in the chest leads, and inverted T in V_5 and V_6 (Fig. 5, *B*).

Summary: The electrocardiogram of a 56-year-old man who suffered from anginal attacks displayed high T waves in the chest leads for three years as the only abnormal sign. Subsequently the high T waves following another attack were replaced by signs of posterior infarction.

CASE 6.—W. S. B., a 59-year-old man, was first seen on May 5, 1948. He gave a four-year history of chest pain on exertion which radiated to the left arm. The physical and fluoroscopic findings were normal, the blood pressure 170/50 mm. Hg, sedimentation rate and white cell count were normal.

The electrocardiogram taken on the same day (Fig. 6, *A*) showed high T waves in the chest leads (CR_3, CR_4, and CR_5), no change in QRS.

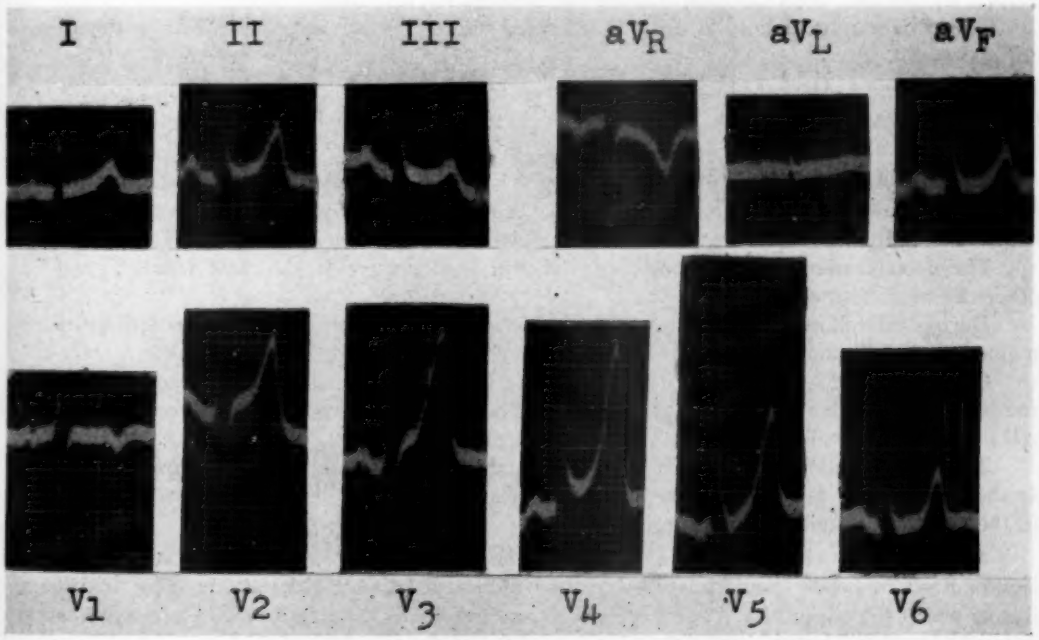


Fig. 7.—Case 8. Very high T waves in the chest leads, V_2 (amplitude 12 mm.), V_3 (amplitude 17 mm.), V_4 (amplitude 19 mm.), V_5 (amplitude 13 mm.). Frequent anginal attacks. Sudden death two weeks later.

One week later the patient suffered a severe precordial pain. The electrocardiogram was obtained about one hour after the attack. It showed a marked depression of RS-T in all limb and chest leads, and the amplitude of the T waves in the chest leads was reduced (Fig. 6, *B*).

The patient died suddenly four days later. An autopsy was not obtained.

Summary: High T waves in the chest leads were the only abnormal electrocardiographic sign in a patient who suffered from anginal attacks. The high T subsequently became replaced by a marked depression of RS-T in the limb and in the chest leads, indicating a subendocardial lesion.

CASE 7.—L. M. H., 55 years of age, began to have retrosternal pain on exertion or excitement. He was first seen on Oct. 3, 1950. The physical and fluoroscopic findings were normal, blood blood pressure 140/60 mm. Hg. The electrocardiogram resembled the tracing in Case 6 and is not reproduced. It showed very high T waves in the chest leads, otherwise no abnormal signs.

The patient was seen repeatedly in the following years and the electrocardiogram remained unchanged.

On Jan. 22, 1954, the patient complained of retrosternal discomfort. The electrocardiogram taken about two hours later showed a marked depression of RS-T in standard and in the chest leads.

The patient died suddenly one week later. An autopsy was not performed.

Summary: The electrocardiogram of a 55-year-old patient who had suffered from frequent anginal attacks during a four-year period displayed high T waves in the chest leads as the only abnormal sign. The high T waves later became associated with the characteristic signs of a subendocardial lesion.

CASE 8.—C. F. was a 41-year-old man who began to have attacks of severe pain in the left arm and left chest in the first week of February, 1954. The pain lasted about thirty minutes and usually occurred after meals or excitement. The patient was seen on March 16, 1954. The heart was not enlarged, the sounds were normal, the blood pressure 120/60 mm. Hg.

The electrocardiogram taken on this day showed strikingly high T waves in the chest leads: V_2, V_3, V_4, and V_5, otherwise no abnormal signs (Fig. 7).

The patient refused hospitalization. He died suddenly two weeks later. An autopsy was not performed.

Summary: A 41-year-old man had suffered from severe attacks of precordial pain. The electrocardiogram showed huge T waves in the chest leads as the only abnormality. The patient died suddenly about eight weeks after the onset of symptoms.

CASE 9.—A. C. W., a man 42 years of age, was well until 1950 when he noticed slight retrosternal pressure after meals or excitement. He was first seen on Sept. 19, 1950. The heart was normal in size and shape, the heart sounds normal, the blood pressure 130/70 mm. Hg, Kahn negative.

The electrocardiogram taken on this day showed high T waves in the chest Leads V_2 and V_3, otherwise no abnormal signs (Fig. 8, *A*).

During the following two years the patient's condition as well as the electrocardiogram remained essentially unchanged.

On Oct. 27, 1952, the patient was admitted to hospital after an attack of intense pressure in the left chest which radiated to the left arm. The blood pressure was 140/60 mm. Hg, white cell count was normal, sedimentation rate 39 mm. per hour.

The electrocardiogram was taken about one hour after the onset of the symptoms (Fig. 8, *B*). It shows a marked depression of RS-T in Leads I and II, decrease in the amplitude of T waves in the chest leads, and slightly negative T in V_6.

Summary: An electrocardiogram taken one hour after the attack showed a marked depression of RS-T in Leads I and II indicating a subendocardial lesion. In the preceding two years, during which the patient had suffered frequent precordial pain on exertion, the electrocardiogram showed very high T waves in the chest leads as the only abnormal feature.

CASE 10.—F. C., a man 58 years of age, gave a history of precordial pain on exertion during the past three months. He was first seen on April 18, 1950.

The physical and fluoroscopic findings were normal, blood pressure 155/80 mm. Hg, white cell count and sedimentation rate normal.

The electrocardiogram showed tall peaked T waves in the chest leads and no change in QRS (Fig. 14, *A*).

Four days later while walking he suffered a severe pain across the chest extending to both arms. The pain lasted about 10 minutes. A few hours later after the dinner he suffered another attack and perspired profusely.

On admission to the hospital on April 24, 1950, there was no essential change in the physical findings, blood pressure was 120/80 mm. Hg, white cell count 8,500, sedimentation rate 57 mm. per hour.

The electrocardiogram (Fig. 14, *B*) showed signs of acute anteroseptal infarction and one week later the high T waves in the chest leads became inverted (Fig. 14, *C*).

The patient died four years later and the autopsy demonstrated an old apical myocardial infarction extending to the interventricular septum.

Summary: A 58-year-old man had suffered frequent anginal attacks on exertion. The electrocardiogram during this period showed tall T waves in the chest leads as the only abnormal sign. After the last attack the high T waves became replaced by the characteristic signs of an

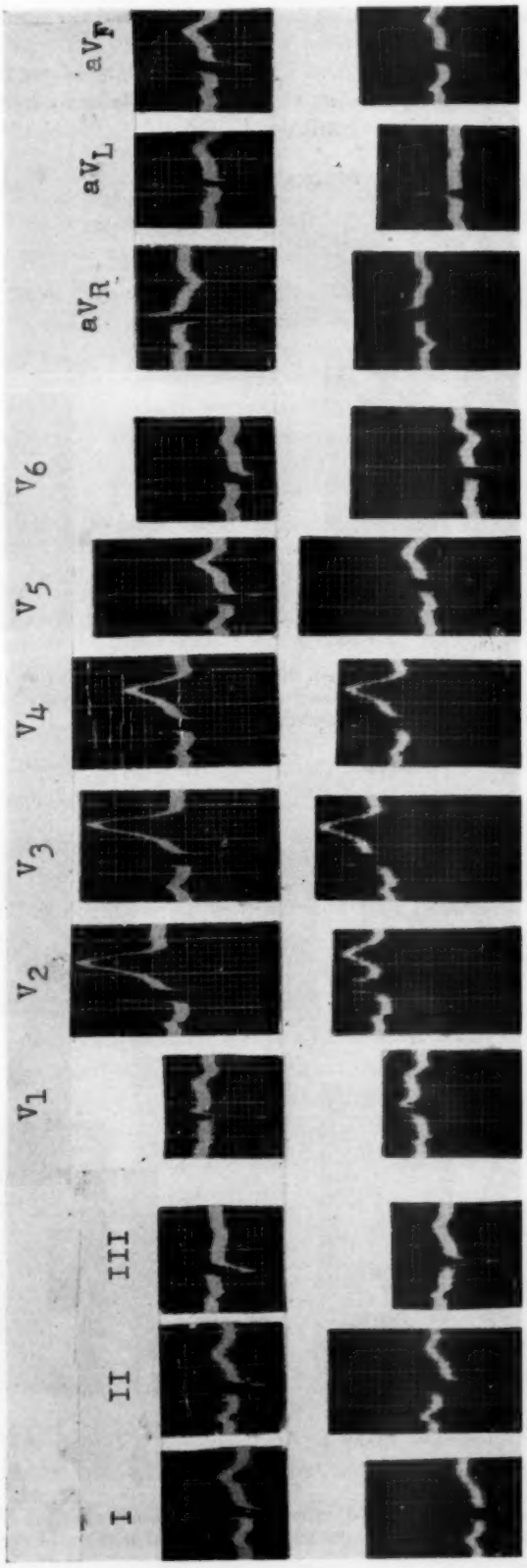


Fig. 8.—Case 9. *A*, Frequent attacks of precordial pain on exertion. High T waves in V_2 (amplitude 13 mm.), V_3 (amplitude 14 mm.), no change in QRS. *B*, obtained one hour after the onset of an attack, shows a marked depression of RS-T in Leads I and II, decrease of R and of the amplitude of T waves in the chest leads, negative T in V_6 and in aV_L.

acute anteroseptal infarction. The patient died four years later and the autopsy confirmed the diagnosis.

CASE 11.—C. R. R. was a 61-year-old man who gave a history of nocturnal dyspnea and precordial pain on exertion during the preceding eight months. He was admitted to hospital on Oct. 29, 1954, with advanced signs of left ventricular failure.

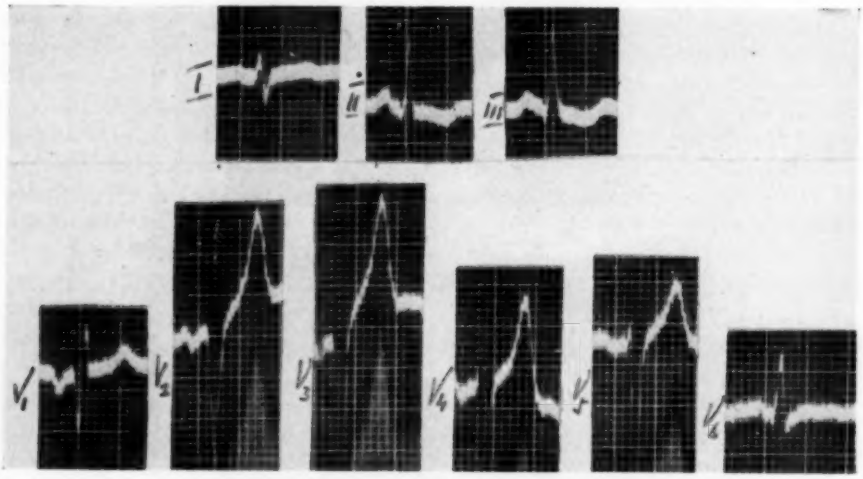


Fig. 9.—Case 11. Tall peaked T waves in V_{2-5}, deep Q in V_1, QS complex in V_{2-5} with small initial R in V_4 and V_5. Diagnostic signs of an old anteroseptal infarction. Autopsy: old fibrous scars of the anterior wall and of the anterior portion of the interventricular septum.

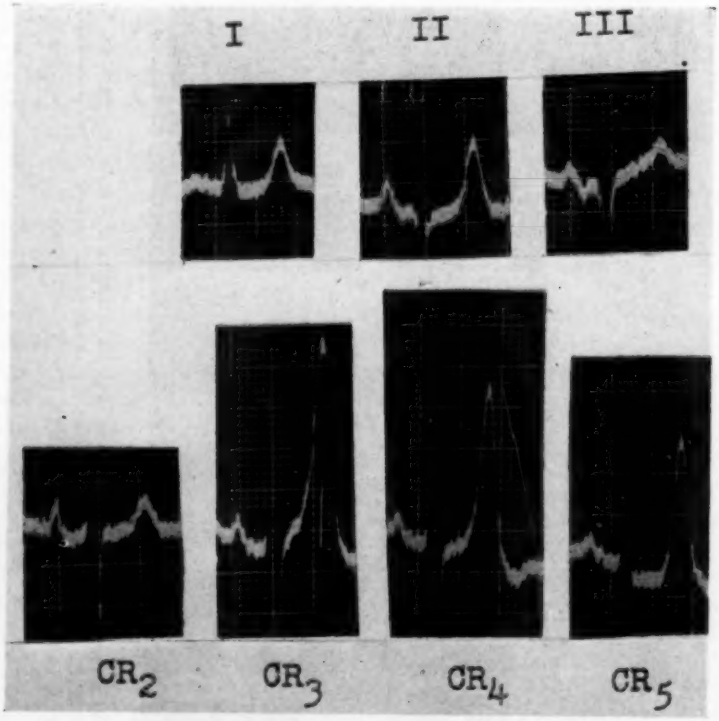


Fig. 10.—Case 12. Huge T waves in CR_{2-5}, otherwise no abnormal changes. Ten-year history of anginal pain. Autopsy: old healed infarction of the basal portion of the septum.

The electrocardiogram (Fig. 9) showed low T in Lead I, depressed R-T in Leads II and III, deep Q in V_1, very high T in chest Leads V_2, V_3, and V_4, and very small R in V_4 and V_5 indicating an old anteroseptal infarction.

The patient died two weeks later and the autopsy revealed a thin anterior wall of the left ventricle and on section old fibrous scars extending to the anterior portion of the interventricular septum.

Summary: The electrocardiogram of a patient who suffered from anginal attacks displayed high T waves in the chest leads associated with signs of anteroseptal infarction. The post-mortem examination confirmed the diagnosis.

CASE 12.—E. McP., a 73-year-old man, gave a ten-year history of anginal pain on exertion and after meals. He was unable to walk more than two blocks due to increasing chest pain. He was admitted to hospital on Oct. 10, 1947. On admission the blood pressure was 120/70 mm. Hg, the heart slightly enlarged to the left, and in the mitral area a loud systolic murmur was audible. The sedimentation rate was 26 mm. per hour, white cell count normal, Kahn negative.

The electrocardiogram (Fig. 10) obtained on this day showed huge T in CR_3, CR_4, and CR_5. The T wave in the limb leads was not strikingly high but sharply peaked and of narrow shape; there was no change in QRS. The patient died two months later and the autopsy revealed an area somewhat smaller than a fifty-cent piece of dense hyaline fibrosis toward the basal portion of the septum, indicating an old healed infarct. The coronary vessels were tortuous, calcified, and the lumina markedly narrowed.

Summary: A 73-year-old man had suffered for 10 years from anginal pain. The electrocardiogram showed huge T in the chest leads and the post-mortem examination revealed an old septal infarct.

DISCUSSION

The electrocardiograms in the cases presented above have been obtained from patients suffering from anginal attacks on exertion.

One hundred were men and ten were women. Their ages ranged from 30 to 70 years. In the majority of cases the symptoms were frequent recurrent attacks of moderate pain or pressure in the retrosternal region radiating to the arms or to the neck, mostly during exertion or after emotional stress. Only in two cases were severe chest pain and profuse perspiration observed. Further, the patients had one common feature: they were emotionally very tense and highly strung. However the blood pressure was not elevated, the heart sounds, sedimentation rate, and white blood cell count were normal.

Large upright T waves in the chest leads and occasionally also in the limb leads were the striking features of the electrocardiogram. In addition to the large amplitude the T waves displayed also a characteristic contour: a sharply peaked wave with symmetrical limbs. The remainder of the electrocardiogram was entirely normal.

The electrocardiograms were taken during the anginal attack or a few hours after the onset of the symptoms and repeated at frequent intervals in order to determine the duration and the course of these tall T waves.

While the normal amplitude of the T wave in the chest leads is considered to be around 8 to 9 mm.,[11,12] in the cases presented here the amplitude of T in V_2 was 10 to 21 mm.; in V_3, 9 to 27 mm.; and in V_4, 9 to 23 mm.

On the basis of the clinical picture as well as the post-mortem findings it was possible to distinguish four different groups among the electrocardiograms with high T waves.

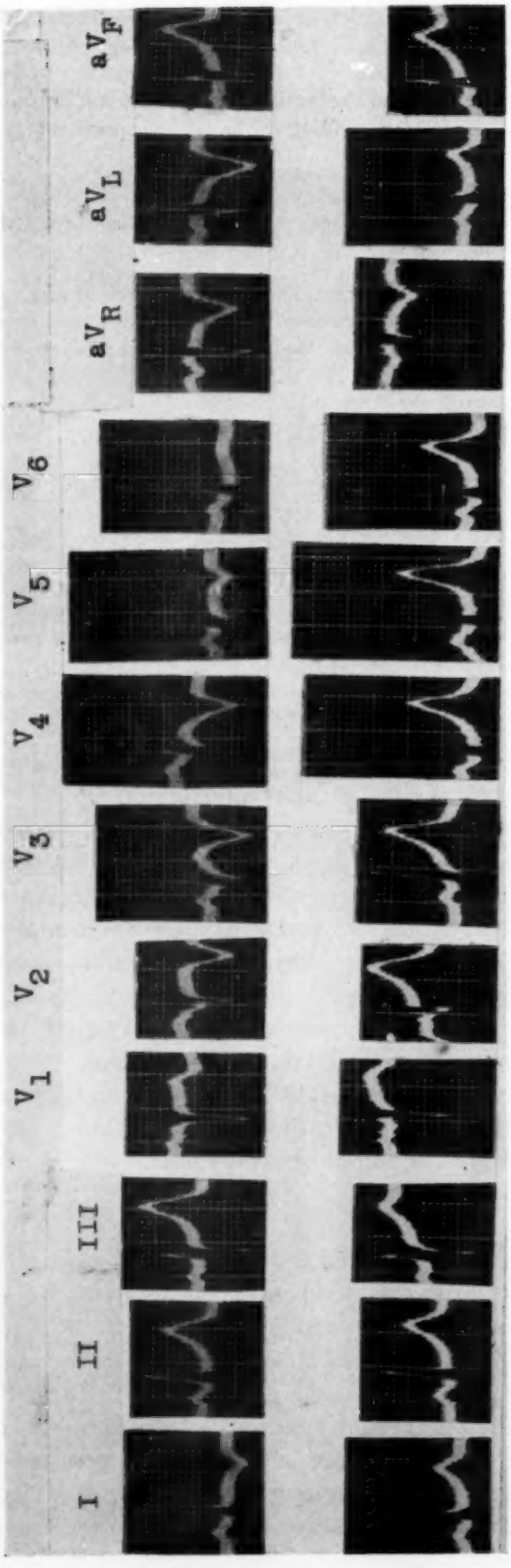


Fig. 11.—*A* (Dec. 27, 1951), signs diagnostic of anteroseptal infarction. *B* (June 24, 1955), four years later, tall peaked T waves in V_{3-6}, otherwise no abnormal changes.

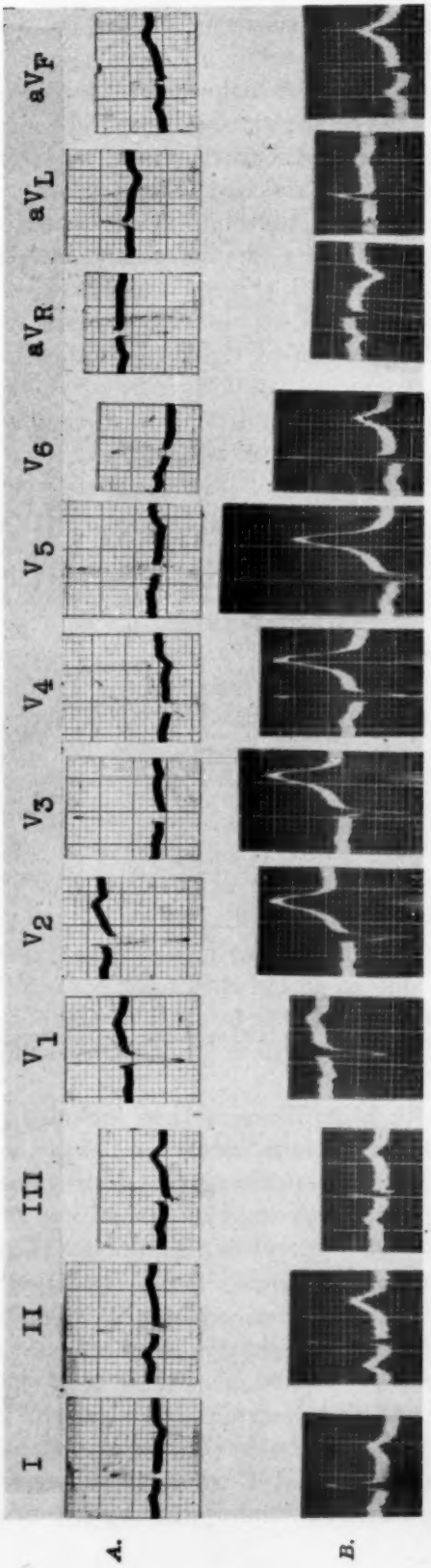


Fig. 12.—*A* (Feb. 25, 1952), obtained one day after a severe attack of precordial pain, signs indicating myocardial ischemia. *B* (June 29, 1954), two years later, tall peaked T waves in V_{2-5}, minute initial R in V_2 and V_3, suggestive of old healed septal infarction.

1. The first group included seventeen patients (15.3 per cent) with frequent anginal attacks. They displayed high T waves mostly in the chest leads, in four cases also in limb leads. These high T waves later disappeared and were followed by characteristic signs of anterior infarction in seven cases, of posterior infarction in three cases, of combined anterior and posterior infarction in one case, and of subendocardial lesion in six cases.

The duration and evolution of the high T waves varied greatly. In two cases they disappeared within twenty-four hours and were followed by an elevation of S-T and later by inverted T (Figs. 1 and 4). In the remaining fifteen patients the high T waves persisted for months or years and were replaced by negative T or by marked depression of RS-T (Figs. 2, 3, 5, 6, and 8). Seven patients of this group died suddenly following another attack. The interval between the onset of the symptoms and death varied between four days and two years. One patient, whose electrocardiogram displayed persisting unchanged high T waves in the chest leads, died suddenly eight weeks after the onset of the symptoms (Fig. 7).

2. The second group included thirty-three patients (30 per cent) who had a history of coronary occlusion two to ten years previously. Their electrocardiograms displayed high T waves which persisted unchanged during observation over a period of four to seven years. The high T waves were associated with the characteristic signs of posterior infarction in fourteen cases (42.4 per cent) and of anterior infarction in fourteen cases (42.4 per cent). In the remaining five cases (15.1 per cent) the high T waves in the chest leads were the only striking sign in the electrocardiogram which otherwise was entirely normal (Fig. 10). In three instances electrocardiograms taken three, four, and seven years previously were available showing the presence of anterior or anteroseptal infarction (Figs. 11 and 12). In two of these cases the post-mortem examination also revealed an old healed septal infarction.

3. The third relatively small group included six patients (5.4 per cent) with severe aortic stenosis due to an old rheumatic endocarditis. The patients had frequent precordial pain on exertion but otherwise no signs of heart failure. Their electrocardiograms showed persistent large T waves during the observation period (Fig. 13).

These three groups with high T waves thus included patients whose predominant symptoms were the anginal attacks. The attacks were either the forerunner of the coronary occlusion or a sign of a previous myocardial lesion. This correlation of the high T waves and the clinical and post-mortem findings justify consideration of the high T waves as a sign of a myocardial lesion.

In animal experiments a relationship between high T and a myocardial injury has been reported. Smith[5] obtained high T immediately after ligation of coronary artery. Wilson and associates[6] pointed out that the T-wave changes are due to transient disturbances which affect the return of the muscle from the active to the resting state and the abnormally tall upright T in epicardial leads indicates that the magnitude of the electrical forces generated late in systole is abnormally great. High precordial T waves have been found after cooling the inner surface of a dog's heart in the apical region, indicating a slowing of

subendocardial repolarization due to an ischemia of the inner wall of the left ventricle.[7-10]

4. In addition to the three groups reported above there was a large number (fifty-four) of patients who had frequent anginal attacks and who had high T waves in precordial leads. They had no history of previous coronary occlusion, the physical and laboratory findings were normal, and during the observation period of one to two years the high T waves persisted unchanged. Two patients in this group died suddenly, one patient eight weeks, another twenty months after the onset of the symptoms.

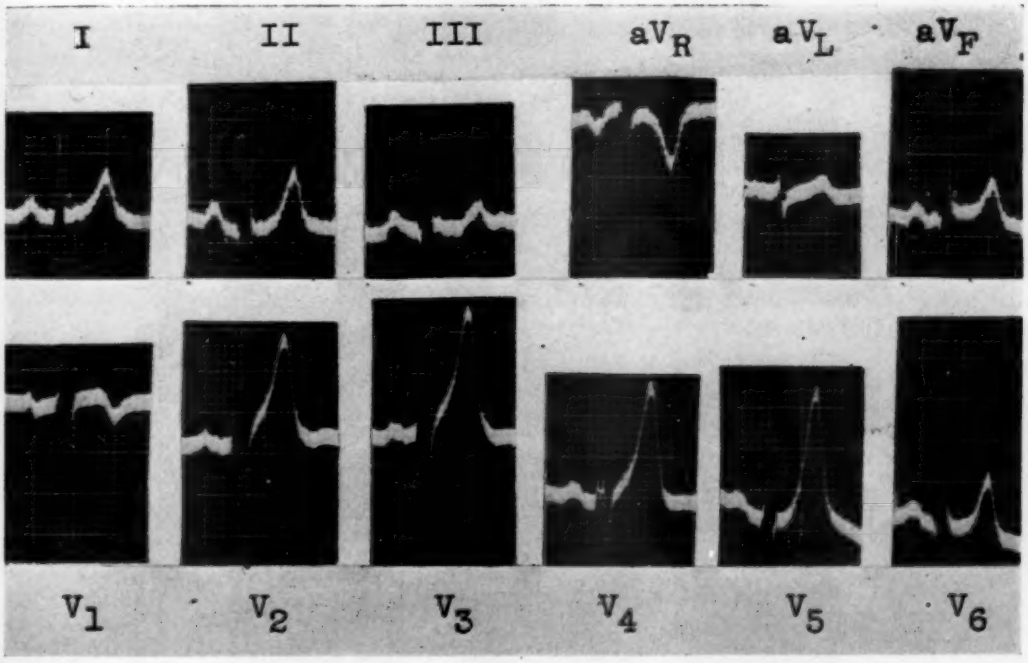


Fig. 13.—Tall peaked T waves in chest leads, otherwise no significant changes. A 35-year-old patient suffering from frequent anginal attacks on exertion. Clinical diagnosis: aortic stenosis due to an old rheumatic endocarditis.

In a few cases a decrease in the amplitude of the high T waves was noted during a prolonged period of abstention from work, during which the patients were free from anginal attacks. A question may arise whether the large T may not be occasionally a normal occurrence. However most of the investigators report that the high T was rarely observed in healthy persons and considered high T of pathologic significance.[11,12]

Sharply peaked tall T waves are also known to occur in uremia as a sign of hyperpotassemia. Only in two patients presented here has a transient increase in potassium level been found. The usual method of potassium estimation involves analysis of venous blood. However it has been pointed out by Da Silva[13] that the tissues remove excess of potassium rapidly from the veins and, in consequence, low serum potassium level, unless determined in arterial blood, may not indicate the true level of potassium.

Fig. 14.—Case 10. *A* (April 18, 1950), frequent severe attacks of precordial pain. Sharply peaked T waves in V_{1-5}. The amplitude of T in V_2 and V_3, 10 mm. *B* (April 24, 1950), diagnostic signs of an acute anteroseptal infarction: elevation of R-T in V_{2-5} and in aVL, QS complex in V_2 and V_3. *C* (May 19, 1950), sharply inverted T waves in chest Leads V_{2-6}.

SUMMARY

One hundred and ten patients with recurrent anginal attacks are reported who displayed very high T waves in the chest leads as the only electrocardiographic abnormality.

On the basis of the clinical as well as of the post-mortem findings four groups of patients were distinguished.

In the first group which included seventeen patients (15.3 per cent) the high T waves preceded the development of the characteristic signs of myocardial infarction.

The second group included thirty-three patients (30 per cent) who had a history of a previous coronary occlusion and the old electrocardiogram displayed signs of infarction. The high T waves persisted in this group up to seven years.

The third group included six patients (5.4 per cent) who had a severe aortic stenosis due to an old rheumatic endocarditis.

In the fourth group of fifty-four patients (49 per cent) the high T waves have persisted and the patients are still under observation. The only change observed in this group was a marked decrease in the amplitude of T in five patients during prolonged rest.

The association of the high T waves with frequent anginal attacks on exertion and the large number (50.7 per cent) of patients in which myocardial damage was proved in follow-up studies point to the pathologic significance of the high T waves.

REFERENCES

1. Wood, F. C., Bellet, S., McMillan, Th. C., and Wolferth, C. C.: Arch. Int. Med. **52**:752, 1933.
2. Bohning, A., and Katz, L. N.: Am. J. M. Sc. **186**:39, 1933.
3. Wood, F. C., and Wolferth, C. C.: Am. Heart J. **9**:706, 1934.
4. Dressler, Wm., and Roesler, H.: Am. Heart J. **34**:627, 1947.
5. Smith, F. M.: Arch. Int. Med. **22**:8, 1918.
6. Wilson, Frank N., Johnston, F. D., and Hill, Ian G. W.: Am. Heart J. **10**:1025, 1935.
7. Bayley, R. H., La Due, J. S., and York, D. J.: Am. Heart J. **27**:164, 1944.
8. Byer, E., Ashman, R., and Toth, L. A.: Am. Heart J. **33**:796, 1947.
9. Hecht, H.: Am. Heart J. **37**:639, 1949.
10. Hellerstein, H. K., and Liebow, I. M.: Am. Heart J. **39**:35, 1950.
11. Stewart, C. B., and Manning, G. W.: Am. Heart J. **27**:502, 1944.
12. Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A.: Am. Heart J. **27**:524, 1944.
13. Da Silva, J. L.: J. Physiol. **108**:218, 1949.

THE EFFECT OF BOUNDARY CONTOUR ON THE DISTRIBUTION OF DIPOLE POTENTIAL IN A VOLUME CONDUCTOR

ROBERT A. HELM, M.D.

CINCINNATI, OHIO

THE potential at a point located on the surface of or within a linear resistive volume conductor containing a single dipole is dependent upon four factors:

1. The strength and orientation of the dipole at the instant of measuring the potential.
2. The distance of the point in question from the dipole center.
3. The resistivity of the conducting medium.
4. The contour of the bounding surface of the volume conductor where it is in contact with a nonconductor such as air.

In accordance with the concept of Burger and van Milaan,[1] the voltage or potential may be represented as the scalar product of two vectors. One of these, representing the strength and orientation of the dipole, is determined by the first factor. The second vector is determined by the three remaining factors. It was originally designated by Burger and van Milaan as the lead vector[1] and subsequently by Frank as the image vector.[2]

It was the purpose of the present study to investigate quantitatively the influence of boundary contour on the lead or image vector. In connection with the factor of medium resistivity it should be stated immediately that inhomogeneity of the volume conductor defies mathematical analysis and can be investigated only to a limited extent experimentally. Burger and van Milaan introduced the factor of inhomogeneity into their experimental work when they constructed their second "phantom" with internal structures of different resistivities.[1] To the author's knowledge, this has been the only attempt to construct a three-dimensional torso model with an inhomogeneous medium although two-dimensional models have been constructed to investigate this factor.[3,4] In the present study the medium will be considered to be homogeneous.

Frank[2] has published a table giving the relative voltages at sixteen points arranged around a transverse level of his homogeneous torso model when a dipole of constant unit strength is consecutively oriented in the right-left or x direction and the anterior-posterior or z direction at seventy-one different locations within the model, each dipole location being situated on the same transverse plane as

From the Cardiac Laboratory, Cincinnati General Hospital, and the Department of Internal Medicine, University of Cincinnati, Cincinnati, Ohio.

Received for publication Feb. 6, 1956.

the sixteen points. In the same publication[2] a formula is given to convert a tabular value to an absolute coefficient expressed in mv./ma.-cm. for a medium with a resistivity of 1000 ohms. When the latter is multiplied by a component of the dipole vector expressed in ma.-cm.,* the product yields millivolts for the potential at the point in question.

Wilson and Bayley[5] have published formulae for the calculation of the electrical field developed when a single dipole exists anywhere in an infinite homogeneous medium and likewise when such a dipole exists anywhere in a homogeneous spherical medium. The formula for the infinite medium can be easily modified to obtain the electrical field of a dipole located in a medium bounded on one side by a single plane of infinite extent.[6] Thus the mathematical tools introduced by Wilson and Bayley may be utilized to calculate data for media with various geometric boundaries. Groups of such data may be individually compared and may also be compared with Frank's experimental findings[2] in order to evaluate the importance of boundary contour.

METHODS

Fig. 9 of Frank's article[2] served as a basis for choosing sixteen points, A through P, arranged around the circumference of a transverse anatomic section of the male torso. The distance of each of these points from the center of the section was carefully measured on an enlarged photograph and converted to centimeters in accordance with the dimensions of the torso section given by Frank. These distances are shown in Fig. 1 on each radius from the center to each of the points. Since the sixteen radii are arranged at 22.5 degree intervals, the x and z components of each of these distances were calculated trigonometrically and will hereafter be referred to as x_p and z_p, respectively.

Four dipole centers at widely different locations were singled out for study. In accordance with Frank's nomenclature,[2] these dipole locations are 00, 80, 08, and 66. The x and z components of the distances of each of these dipoles from the center of the anatomic section were read directly from Frank's Fig. 9 and are indicated here in Fig. 1. Such dipole components will hereafter be designated as x_d and z_d.

It was decided that the values of x_p and z_p for each of the sixteen points and the values of x_d and z_d for each of the four selected dipoles should remain constant for all of the boundary conditions postulated in this study. Furthermore, it seemed desirable that each of the sixteen points should always be located *on* rather than *within* a boundary surface since, in electrocardiography and vectorcardiography, electrodes are usually placed on the body surface rather than within internal organs. To maintain a constant relationship between a given dipole location and each of the sixteen boundary points regardless of boundary configuration† requires that any boundary which has a contour other than that

*The dipole magnitude may be expressed as the product of its strength in millivolts and its pole separation in centimeters. When the orientation of its poles is taken into account a dipole takes on the properties of a vector.

†This requirement does not permit the method illustrated in Fig. 3 of Frank's paper[2] to be utilized. With Frank's method of approaching the problem, the distances between the dipole center and each of the sixteen boundary points are not the same in the sphere as they are in the torso model.

of Frank's torso model must be altered as the voltage at each consecutive point is determined. This method of treatment may be made more lucid by a consideration of the actual boundary conditions selected. These are as follows:

1. An infinite medium without a boundary.

2. A single infinitely large plane. Such a plane is postulated to exist consecutively at each of the sixteen points, A through P, when the voltage of that particular point is calculated as illustrated in Fig. 1. Such a plane is always perpendicular to the radius from the center of the section to the point in question.

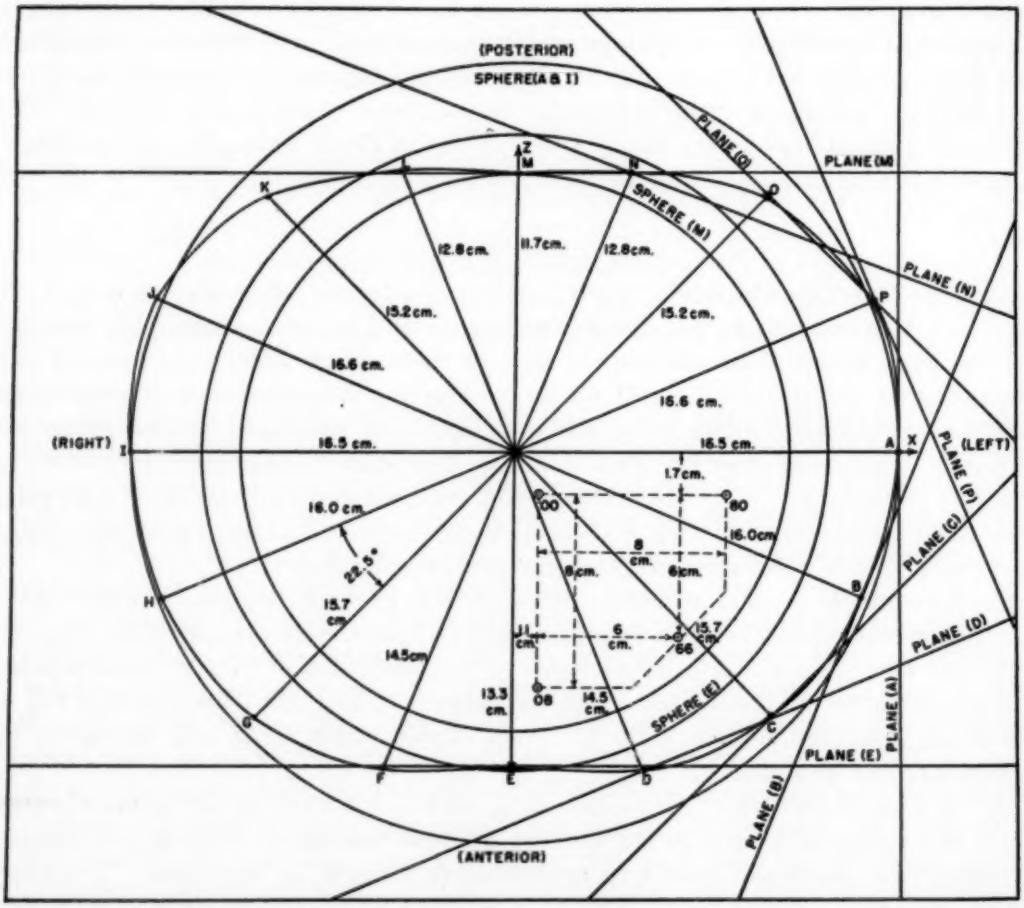


Fig. 1.—Anatomic cross section of thorax demonstrating the positions of the sixteen surface points, *A* through *P*, and the four dipole locations, 00, 80, 08, and 66. The method of replacing the torso surface with a planar or spherical surface at each of the sixteen points is illustrated for a limited number of such points and is described fully in the text.

(In Fig. 1, the locations of nine planes, one for each point, M through E, are illustrated, the seven planes for points F through L being omitted to avoid confusing details.) It should be stressed that only one of these sixteen planes exists at any time, depending upon the point whose voltage is being determined.

3. A single sphere. Such a sphere is postulated to exist consecutively at each of the sixteen points, A through P, when the voltage of that particular

point is calculated. As illustrated in Fig. 1, the sphere always assumes a radius equivalent to the distance of the point in question from the center of the section. In Fig. 1, the locations of three spheres are illustrated, others being omitted to avoid confusing details in the drawing. Thus one sphere for point M and another larger sphere for point E are shown. The location of a third sphere, which serves for both points A and I, is also illustrated. As with points A and I all of the remaining twelve points may be grouped into pairs, each point of a pair being located on a single sphere with a radius equivalent to the distance of the center of the section from each point of the pair. It should be emphasized that only one of these nine spheres exists at any one time, depending upon the point or pairs of points whose voltages are being determined.

4. The boundary contour of Frank's torso model, which, of course, passes simultaneously through each of the sixteen points located on its surface.

It is to be noted that, for all four boundary conditions, the medium is three-dimensional. This cannot be well depicted in a two-dimensional illustration such as Fig. 1 which represents a transverse section.

Calculation of the voltage at each of the sixteen points, A through P, was carried out for each of the first three boundary conditions, when the dipole center was located consecutively at each of the four selected sites and when the dipole axis was first oriented parallel to the x axis and then parallel to the z axis. Thus, the x and z components of the voltage at each of these points were calculated. These components will hereafter be designated as V_x and V_z. The methods of determining such components for each boundary condition are as follows:

1. Infinite medium. Equation (17) of Wilson and Bayley[5] may be modified to yield values in mv./ma.-cm. for a medium with a resistivity of 1000 ohm-cm. At the same time their Equation (17) may be simplified by utilizing the symbols previously defined.

$$\text{Let } x = x_p - x_d, \text{ and}$$

$$z = z_p - z_d$$

Then the final equations are:

$$V_x = \frac{250x}{\pi (x^2 + z^2)^{3/2}} \tag{1}$$

$$V_z = \frac{250z}{\pi (x^2 + z^2)^{3/2}} \tag{2}$$

2. Medium bounded by a single plane. The mathematical treatment of such a condition has been greatly simplified by Nelson's recent discussion[6] of electrical images. In accordance with this concept, the values for V_x and V_z, calculated from Equations (1) and (2), are merely doubled.

3. Medium bounded by a single sphere. Equation (19) of Wilson and Bayley[5] is utilized except that it is modified by replacing the term M by the quantity, $250/\pi$, to yield values in mv./ma.-cm. for a medium with a resistivity of 1000 ohm-cm. For V_x, the direction cosine of the dipole axis with the x axis is 1 and the direction cosines of the dipole axis with the y axis and with the z

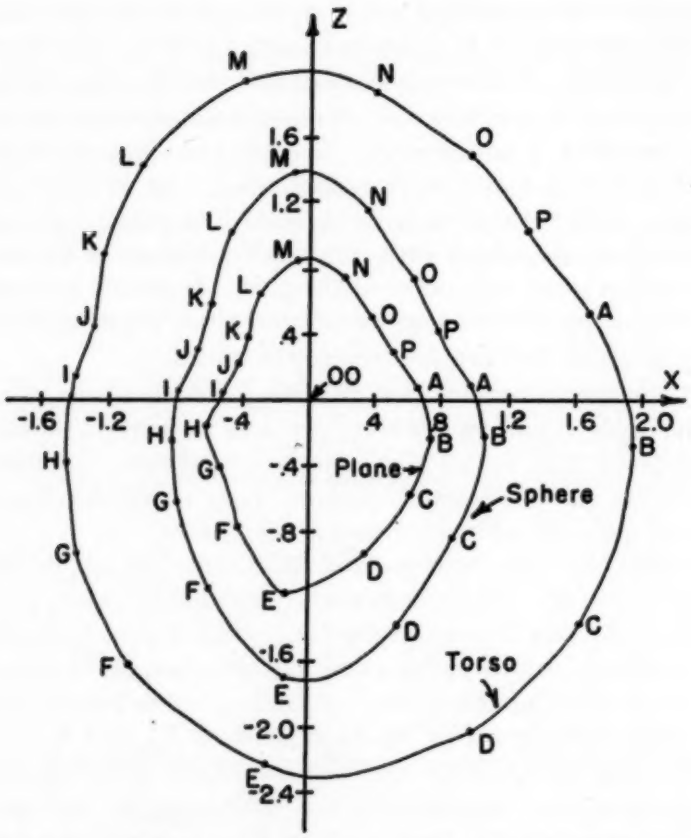


Fig. 2.—Image loops for planar, spherical, and torso boundaries obtained with a single dipole at the *00* location. See text. (The scale of this and subsequent illustrations is in mv./ma.-cm.)

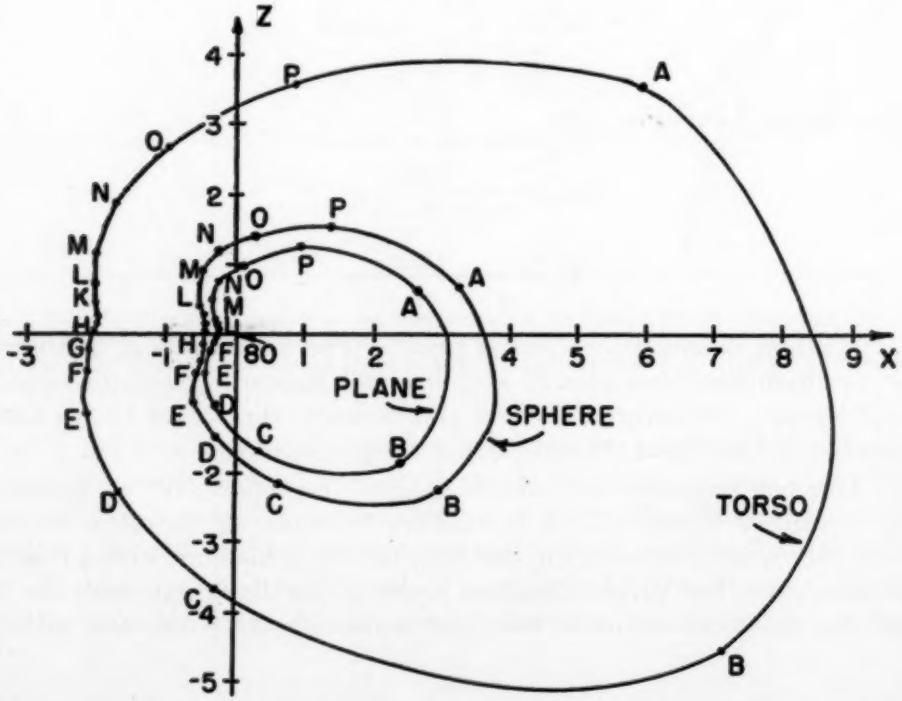


Fig. 3.—Image loops for planar, spherical, and torso boundaries obtained with a single dipole at the 80 location. See text.

axis are each 0. For V_z the direction cosine of the dipole axis with the z axis is 1 and the two remaining direction cosines are each 0. Equation (19) of Wilson and Bayley[5] is more complicated than their Equation (17) and its application is much more time-consuming. Moreover, Equation (19) cannot be simplified in the manner which their Equation (17) was modified (see method 1 above) by the use of the terms herein defined. Rather, the author found it necessary to

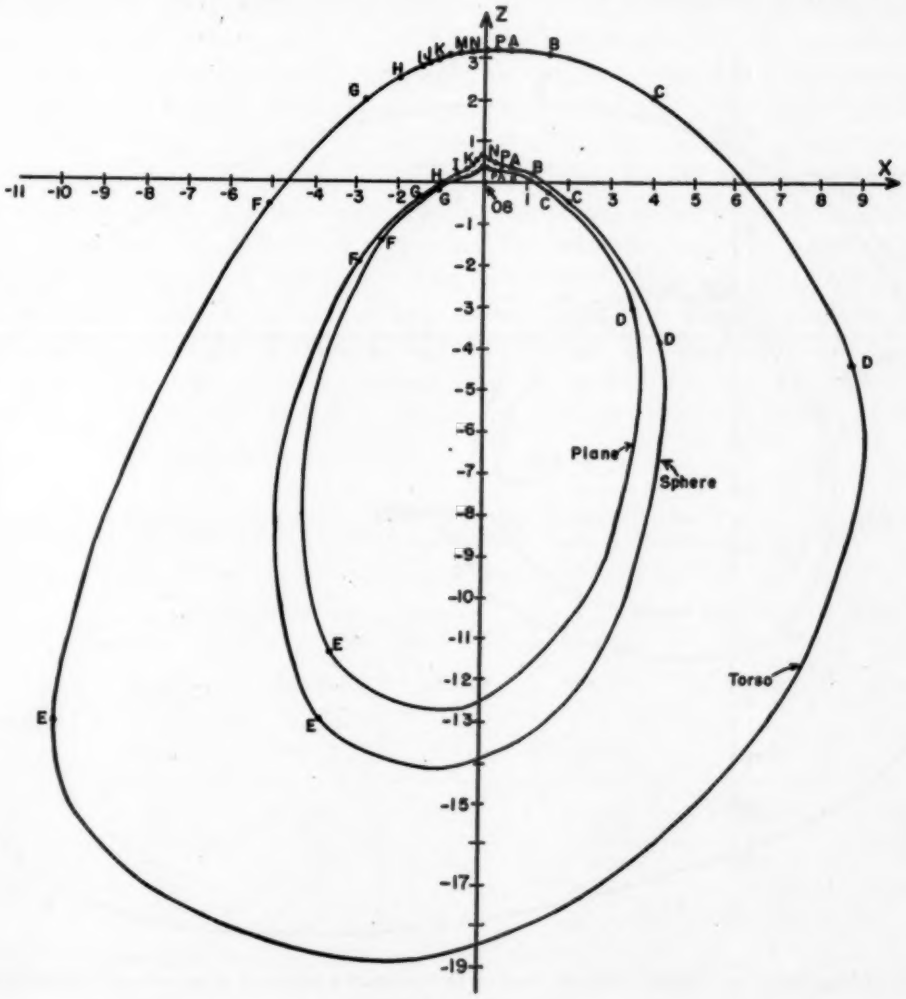


Fig. 4.—Image loops for planar, spherical, and torso boundaries obtained with a single dipole at the 08 location. See text.

convert these terms to the symbols used by Wilson and Bayley to carry out the computations as expeditiously as possible.

4. Medium bounded by the configuration of Frank's torso model. The data given in Frank's Table I for the four selected dipoles were converted to mv./ma.-cm. for a medium with a resistivity of 1000 ohm-cm. by use of the formula given on page 691 of Frank's article.[2]

GRAPHIC RESULTS

The results of the calculations described under Methods are depicted in the form of image loops in Figs. 2 to 5, each figure representing a different dipole location. Since the image loop for the infinite medium is of identical form but half the magnitude of the image loop for the plane-bounded medium, the former is omitted in all of the illustrations. For all four dipole locations the image loop of the spherical medium is consistently somewhat larger than the image loop

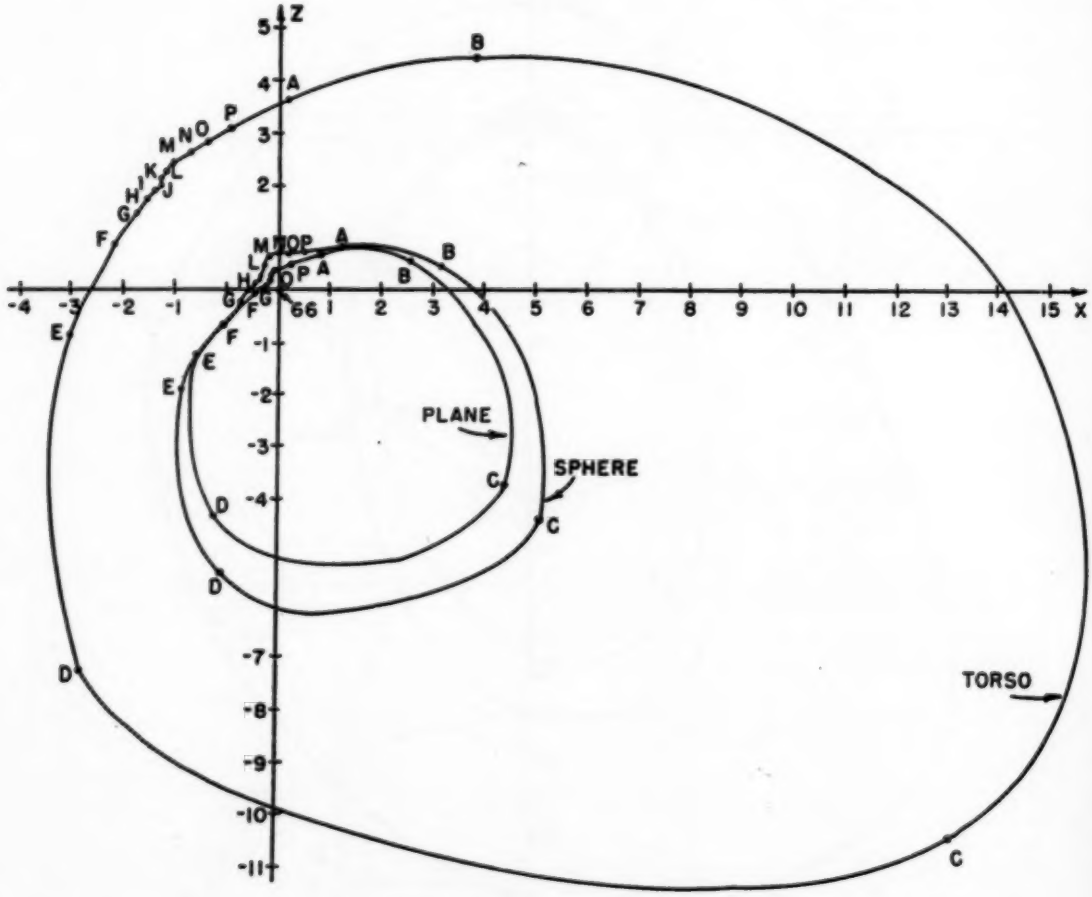


Fig. 5.—Image loops for planar, spherical, and torso boundaries obtained with a single dipole at the 66 location. See text.

of the plane-bounded medium and is, in turn, considerably exceeded in magnitude by the image loop obtained from Frank's torso data. It should be noted, however, that, for any given dipole location, the image loops of the variously bounded volume conductors have a rather similar form. This similarity will be studied quantitatively in the next section. It is likewise apparent that the image loops have widely varying contours as well as varying magnitudes with different dipole locations. Finally, it should be pointed out that, with increasing dipole eccentricity, the distances in image space between points which are anatomically rather close to the dipole, increase tremendously. (See especially Figs. 4 and 5.)

This introduces a large error into the construction of image loops from measurements taken at a limited number of surface points of a torso model. A practical method of surmounting this difficulty will be described in the discussion.

STATISTICAL RESULTS

Correlation coefficients were calculated for the x components and for the z components of the sixteen image vectors of paired volume conductors with different boundary configurations. These paired combinations are plane and sphere, plane and torso model, and sphere and torso model. (The results with the unbounded infinite medium were omitted from the correlations because of their simple linear relation to the results with the plane-bounded medium.) These correlation coefficients are listed in Table I. All are extremely high, approaching unity, and are very highly significant in view of the values of r, given in the legend of Table I, necessary for achieving various levels of probability for 14 degrees of freedom. The best correlations are those between the plane-bounded and spherical volume conductors, but the correlations of the torso model with each of these is sufficiently high to indicate that, for a given dipole location, the results obtained from measurements on the torso model could be accurately predicted by means of regression from data calculated for a much simpler boundary condition.

TABLE I. CORRELATION COEFFICIENTS (r) BETWEEN POTENTIALS OF POINTS LOCATED ON THE SURFACES OF VOLUME CONDUCTORS HAVING VARIOUS BOUNDARY CONTOURS*

DIPOLE LOCATION	x COMPONENT OF IMAGE VECTOR			z COMPONENT OF IMAGE VECTOR		
	PLANE SPHERE	PLANE TORSO	SPHERE TORSO	PLANE SPHERE	PLANE TORSO	SPHERE TORSO
00	.999	.995	.996	.999	.992	.992
80	.996	.987	.982	.991	.978	.959
08	.996	.994	.988	.999	.984	.990
66	.996	.975	.966	.997	.970	.956

For 14 degrees of freedom, $r_{.05} = .497$, $r_{.01} = .623$, and $r_{.001} = .742$.
*See text.

The influence of varying dipole eccentricity on the correlation coefficients was next investigated by means of analysis of covariance. The object of such an analysis was to determine whether the slope of a regression line between the image vectors, obtained with any given pair of differently bounded volume conductors, is dependent upon dipole location. The values of F, obtained for the six separate analyses of covariance designed to answer this question, are listed in Table II. The tabular values[7] of F required to attain various levels of probability are given in the legend of this table. Examination of Table II indicates that, with the exception of the x components of the image vectors of

the plane-bounded medium and the torso and of the spherical medium and the torso, the average variation between the regressions for the different dipoles with 3 degrees of freedom is significantly greater than the average variation among individual regressions with 56 degrees of freedom. One may conclude that neglect of a consideration of dipole location significantly reduces the accuracy of applying regression equations in predicting the image vectors obtainable with one volume conductor from those calculated with another conductor having a different boundary contour.

TABLE II. VALUES OF VARIANCE RATIOS (F) OBTAINED FROM ANALYSIS OF COVARIANCE FOR TESTS OF SIGNIFICANCE OF DIFFERENCES IN REGRESSIONS FOR FOUR DIPOLE LOCATIONS*

X COMPONENT OF IMAGE VECTOR			Z COMPONENT OF IMAGE VECTOR		
PLANE SPHERE	PLANE TORSO	SPHERE TORSO	PLANE SPHERE	PLANE TORSO	SPHERE TORSO
6.56	1.90	1.37	11.68	31.16	30.48

For 3 and 56 degrees of freedom, $F_{.05} = 2.78$, $F_{.01} = 4.17$, and $F_{.001} = 6.26$.
*See text.

DISCUSSION

If a conducting medium is infinite in extent, the isopotential lines developed by a dipole are circular and are always perpendicular to the lines of current flowing between the poles. If the volume conductor is not infinite, its boundaries modify such isopotential lines and current lines. The outermost isopotential lines are perpendicular and the outermost current lines are parallel to the adjacent boundary surface. Isopotential and current lines located in positions which are intermediate between the boundary and the dipole are also modified in their directions but to a lesser extent. Isopotential and current lines in the immediate vicinity of the dipole and relatively remote from the boundary surface are only slightly altered. Throughout the electrical field isopotential lines always remain perpendicular to the lines of current flow regardless of the boundary contour. On the basis of these facts, it is evident that boundary contour must influence potential differences measured on the body surface. The investigation reported herein was designed to determine the magnitude of this effect and, if possible, to devise statistical methods which would permit one to take the boundary factor into consideration in the construction of leads for an accurate vectorcardiographic reference frame.

Since, for practical reasons, electrocardiographic and vectorcardiographic measurements involve body surface potentials, it did not seem pertinent to include an investigation of the effect of boundary contour on the potentials at points within the conducting medium. It was for this reason, together with the necessity of avoiding the effect of varying dipole eccentricity by maintaining a constant relationship between given dipole locations and surface points, that the author chose to assume, whenever necessary, a separate boundary for each point whose potential was to be determined. This is admittedly an artificial

concept but one which isolates the effects of those factors enumerated in the introduction, which together determine the image vector.

The introduction of a single plane into an infinite medium increases the potential developed by a dipole at a point, and, if the point in question is located on the plane, its potential is doubled.[6] Introduction of the dipole-containing medium into a sphere also increases considerably the potential of a point located on its surface; in almost all instances the sphere has a greater effect than the plane on image vector magnitude. One would expect that the introduction of a boundary having the configuration of the human torso would also increase the potential of a point on its surface but that this increase would be of the same order of magnitude as that produced by the introduction of the plane or sphere. The explanation for the consistently and rather markedly increased voltages which Frank obtained experimentally in comparison with those which are calculated with a plane-bounded or spherical medium is not clear to the author. The deviation of the properties of Frank's experimental dipole from those of a mathematical dipole would not appear to be an adequate explanation if one examines an equation derived specifically to define such a deviation.[5] The fact that Frank's torso potentials correlate so closely with the calculated potentials of the other boundaries indicates that any error which could conceivably exist between experimentally and theoretically determined values must at least be a systematic one.

From a study of Figs. 2 to 5 it appears that boundary contour rather profoundly effects the magnitude of dipole potentials. However, for a given dipole location, or in the broader sense, for a given degree of dipole eccentricity, it is possible to deal with the effect of boundary contour by means of the regression technique. This is of considerable practical importance if an accurate image loop for a dipole on the transverse plane of the torso model is desired. For example, it is evident that the curved lines joining points D and E in Fig. 4 or points B and C in Fig. 5 must represent very gross approximations of these segments since there are no intermediate points to determine their true courses. Any number of such points, however, could be rather accurately determined by the general technique to be described. This technique will be illustrated in detail for the 23 dipole location since such a site represents a typical position of the electrical center of the heart during the inscription of the QRS complex[2] and, as such, the regression equations given are useful for problems involving the concept of a ventricular electrical center.

Since the correlation coefficients between the plane-bounded medium and the torso model are certainly as high as those between the spherical medium and the torso model, the time-consuming nature of the calculations involved in determining the potentials of points on the sphere represents a deterrent to the use of the latter. Moreover, since the potentials of points on a plane bear a simple 2:1 relation to those of points in an infinite medium, there is no diminution in the accuracy of a regression if it is based on an infinite volume conductor rather than on a plane-bounded medium. Equations (1) and (2), given earlier, may, therefore, be used to determine for an infinite medium the x and z components of the potentials developed at points A through P by a dipole at the 23

location whose x and z components of eccentricity are 3.1 and 4.7 cm. respectively. If these component potentials are then taken in conjunction with Frank's coefficients for these points, expressed in mv./ma.-cm. for the 23 location, the following correlation and regression coefficients may be calculated:

Potential Component	r	b
x	.994	4.904
z	.988	4.007

The appropriate regression equations are:

$$\overline{V}_x = 4.904\ V_x - .208 \tag{3}$$

$$\overline{V}_z = 4.007\ V_z + .431 \tag{4}$$

where $\overline{V}_x$ and $\overline{V}_z$ are the predicted x and z components of the potential of a point on the torso estimated from the voltage components, V_x and V_z, calculated for a corresponding point in an infinite medium.

As a practical example of the use of these equations let us consider points C and D whose radii on the anatomic transverse plane intersect at an angle of 22.5 degrees. Within this angle let us construct eight additional radii from the center of the cross section to the boundary in such a way that the ten radii from C to D now form equal angles at 2.5 degree intervals. The lengths of these additional radii may be measured on an enlarged photograph of Fig. 9 of Frank's paper[2] and their respective x and z components, i.e. x_p and z_p, may be determined trigonometrically. The x and z components of the potentials developed in an infinite medium at each of the eight points where the additional radii intersect the anatomic boundary are then calculated by means of Equations (1) and (2). The eight values of V_x and the eight values of V_z are next substituted in Equations (3) and (4) and $\overline{V}_x$ and $\overline{V}_z$ are determined for each of the new radii. Plotting the paired values of $\overline{V}_x$ and $\overline{V}_z$ supply eight additional points for the predicted segment of the image loop between C and D for the 23 location of the dipole in Frank's torso model. These points fall along a curved line which is determined by the regression involving all sixteen points, A through M. However, because of errors within the regression they cannot be expected to fall upon the true torso image segment between C and D. A local correction for regression error in the region between C and D may be carried out by interpolation. Let us consider the following values for points C and D:

Potential Component	Frank's Experimental Value	Regression Value	Correction Ratio
x for point C	2.875	2.694	1.067
z for point C	−2.078	−1.465	1.418
x for point D	1.243	1.087	1.144
z for point D	−3.395	−3.325	1.021

The x and z components of the potentials of the points between C and D may be corrected by multiplying the regression values by the appropriate combinations of the correction ratios. For example, the x and z correction factors for the point which is 15 degrees from C and 7.5 degrees from D are 1/3 (1.067) + 2/3 (1.144) = 1.118 and 1/3 (1.418) +2/3 (1.021) = 1.153, respectively. Such an estimate, based upon both regression and interpolation, is much superior to an estimate made by either technique alone. Regression gives full weight to the general contour of the entire image loop in determining the position of an image point between C and D. Interpolation provides an appropriate correction for local regression error. If interpolation were applied as the sole method of determining the locations of intermediate torso points in image space, marked errors would result since all such intermediate points would be located on a straight line between the two points where voltage measurements were taken.

A detailed description of this method of predicting the position of intermediate points in image space has been given because it permits Frank's published data to be applied to the estimation of the image vectors of leads, the electrodes of which are applied to large segments or areas of the body torso[8,9] rather than at single points or combinations of the points, A through P. Such lead vectors are now being determined in this laboratory to define the characteristics of a spatial vectorcardiographic reference frame which is theoretically more accurate than any in present use and which, at the same time, is sufficiently simple for clinical application.[10]

Of the other two factors (in addition to boundary contour) which influence the magnitude and direction of a lead vector, dipole location is of prime importance. The factor of medium resistivity may be easily taken into account, if the volume conductor is homogeneous, because the calculated or measured potentials at all points are directly proportional to the magnitude of this factor. The work of Schwan and his associates[11] is particularly important since they demonstrated that the resistivity of various tissues of living dogs ranges from 900 (liver) to 1100 (lung) ohm-cm. with coefficients of variation of 8 to 15 per cent. Therefore, the conversion of Frank's measurements to coefficients for a homogeneous medium with a resistivity of 1000 ohm-cm. may permit the application of such data to the human torso without significant error.* Variations of dipole location greatly alter the size and contour of the image loop of the transverse torso section as graphically demonstrated by a comparison of Figs. 2 through 5. However, in previous publications[8,9] the author has described methods of constructing leads which are relatively insensitive to changes in the electrical center of the heart, or preferably, expressed in terms of a more universal concept, leads which possess relatively uniform lead vectors for all points within the myocardium where electric current is generated.[12] In these publications[8,9] tentative proposals for electrode positions and standardizing factors for a vectorcardiographic reference frame were made. The present investigation, dealing primarily with the effect of boundary contour, may provide a method which will allow much more concrete proposals concerning such leads to be made.

*This statement neglects the relatively low resistivity of blood. The effect of the intracardiac blood volume on the application to the human thorax of coefficients obtained with the homogeneous torso model is not clear at the present time.

SUMMARY

The effects of dipole eccentricity and medium resistivity have been isolated from those of boundary contour in such a manner that the effect of variations of the latter on the dipole potentials developed at any given point may be studied in pure form. It was demonstrated that boundary contour significantly alters such potentials but that a high degree of correlation exists between potentials obtained with various boundary configurations if the location of the dipole center remains constant. A method which utilizes regression and interpolation is described for the accurate estimation of the potentials of intermediate points on the transverse plane of Frank's homogeneous torso model. The application of this method should permit the electrode positions and standardizing factors of a vectorcardiographic lead system, described by the author in previous publications, to be determined with greater accuracy.

I wish to thank Mr. Harold Perlman for performing the calculations involved in the application of the Wilson-Bayley equations.

REFERENCES

1. Burger, H. C., and van Milaan, J. B.: Brit. Heart J. **8**:157, 1946; **9**:154, 1947; **10**:229, 1948.
2. Frank, E.: Am. Heart J. **49**:670, 1955.
3. McFee, R., Stow, R., and Johnston, F. D.: Circulation **6**:21, 1952.
4. Brody, D. A., and Romans, W. E.: Am. Heart J. **45**:263, 1953.
5. Wilson, F. N., and Bayley, R. H.: Circulation **1**:84, 1950.
6. Nelson, C. V.: Circulation Research **3**:236, 1955.
7. Fisher, R. A., and Yates, F.: Statistical Tables, New York, 1949, Hafner Publishing Company.
8. Helm, R. A.: Am. Heart J. **50**:883, 1955.
9. Helm, R. A.: Am. Heart J. **52**:323, 1956.
10. Helm, R. A.: An Accurate Lead System for Stereovectorcardiography and Spatial Electrocardiography, Am. Heart J. To be published.
11. Schwan, H. P., Kay, C. F., Bothwell, P. T., and Foltz, E. L.: Fed. Proc. **13**:131, 1954.
12. Helm, R. A.: Am. Heart J. **49**:135, 1955.

THE EFFECT OF BACKGROUND NOISE ON CARDIAC AUSCULTATION

DALE GROOM, M.D.

CHARLESTON, S. C.

WITH THE TECHNICAL ASSISTANCE OF OREN HERRING, B.S., WOFFORD FRANCIS, AND GIBSON SHEALY, B.S.

IT IS axiomatic that to hear a faint cardiac murmur one should listen in a quiet environment. In the average busy hospital, clinic, or office environment there is a more or less constant level of background noise which, though we become acclimated and somewhat oblivious to it, does interfere with auscultatory examination. Particularly, faint heart murmurs, those which often are of the greatest importance in early diagnosis of heart disease and which reach the ear in intensities far lower than those of ordinary conversation and the other sounds to which the ear is more accustomed, are readily masked by extraneous room noises. This masking effect is well known to clinicians. Perhaps less well known is the degree to which the background noise levels actually present in hospital wards and examining rooms can impair one's ability to hear heart murmurs through the stethoscope.

The purpose of this study was to measure the degree of this impairment under conditions simulating those of ordinary cardiac auscultation. We have been unable to find in the medical literature references to any study of this aspect of auscultation.

METHOD AND PROCEDURE

A survey of ambient noise levels present in various rooms of the hospitals and outpatient clinics of the Medical College of South Carolina was made with a sound level meter (General Radio 1551-A) according to accepted acoustical methods. In all cases averages of several decibel* readings were taken under conditions as representative as possible of those obtaining during routine examination of patients. Unusual noises such as the rumble of passing trucks, radios

---

From the Department of Medicine, Medical College of South Carolina, Charleston, S. C.

This study was supported in part by grants from the South Carolina Heart Association and the National Heart Institute.

Received for publication Feb. 4, 1956.

*The unit commonly used to measure intensity, the decibel (db), is in reality a logarithmic expression of the energy required to produce the sound. A 10 db increase in intensity (power) denotes 10 times the intensity, 20 db 100 times, and 30 db 1,000 times the intensity. The scale of decibels used in measuring sound intensities with the sound level meter is based on 0 db (0.0002 dynes per square centimeter sound pressure) as the threshold of normal human hearing.

The ear also is logarithmic as regards intensity, a 1 db change producing a barely perceptible change in loudness of a tone in ordinary physiologic usage.

playing, crying babies, or the clatter of dishes or trays were excluded from the readings in so far as possible. The results of this survey together with readings representative of other familiar environments are listed in Table I.

TABLE I. MEASURED BACKGROUND NOISE LEVELS IN VARIOUS AREAS OF THE HOSPITALS AND OUTPATIENT CLINICS OF THE MEDICAL COLLEGE OF SOUTH CAROLINA

	NOISE LEVEL DECIBELS (r.e. 0.0002 MICROBAR)
Threshold of pain*	130
Inside DC-6 airliner*	105
Boiler room, Medical College Hospital (air conditioning equipment running)	100
Pediatric Clinic examining room	75
Outpatient Clinic waiting room	72
Medical ward, Roper Hospital	70
Heart Clinic office	69
Library	68
Obstetrical Clinic examining room	66
Sound-proofed room with noise channel running	65
Surgicial ward, Roper Hospital	65
Emergency Room, Roper Hospital	62
Private room, Medical College Hospital	60
Medical Clinic examining room	55
Private room, unoccupied floor of Medical College Hospital	40
Sound-proofed room, quiet	35
House in country*	30
Rustle of leaves in gentle breeze*	10
Threshold of hearing*	0

Standard procedure (Am. Standards Assoc. Z24.3-1944)[1] for measuring noise as the ear hears it involves the use of three alternate frequency response characteristics in the sound level meter. These are designated as "weighting networks" A, B, and C, and they are used to selectively discriminate against low-and-high-frequency sounds in accordance with certain "equal loudness" contours (the Fletcher-Munson curves) of human hearing. High intensity sounds are measured on the C scale which has a flat frequency response and records actual sound pressure; those sounds ranging from 55 to 85 db are measured on the B scale, and levels below 55 db on the A scale. All the above noise levels were determined according to this procedure. Values for sound pressures in the sound-proofed room, not shown, are given in the text.

***Taken from the tables of other surveys.[1,2]**

One of the outpatient clinic examining rooms was remodeled with simple, relatively inexpensive sound-proofing measures to exclude extraneous noise. In a room-within-a-room arrangement, inner walls and ceiling were constructed

of acoustical tile of the perforated fiber type having a specified noise reduction factor of 0.65. The door was heavily padded, the window closed off, and a thick carpet placed on the floor. Ambient room noise was thereby reduced to the 35 decibel level shown in Table I, which is in terms of subjective "loudness"—that is, as the ear hears it. Total sound pressure, which includes low frequency rumble and inaudible vibrations, was reduced to 53 db. Although this is not a particularly low noise level on acoustical standards it can be seen from the table to measure vastly lower than other areas where patients are regularly examined. The necessary electronic equipment was placed within this quiet room where the experiment was carried out.

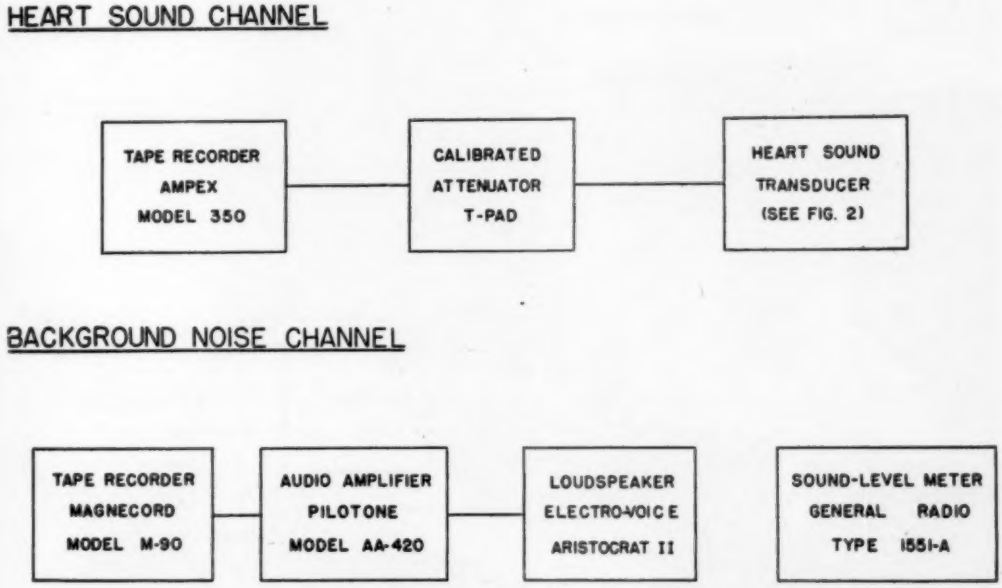


Fig. 1.—Block diagram illustrating arrangement of apparatus into two channels: One for reproduction of the heart sounds and murmur, the other to reproduce background noise in the room. Intensity of the latter was adjusted to a 65 decibel level at the subject by means of the sound-level meter.

In order to reproduce heart sounds at variable intensity for stethoscopic auscultation at different levels of background noise, two sound channels were used which are illustrated in the block diagram of Fig. 1. The first channel reproduced the heart sounds and diastolic murmur recorded on a continuous tape loop from a case of syphilitic aortic regurgitation. This was fed through a calibrated volume control to the heart sound transducer shown in Fig. 2. The transducer was devised from a magnetic loud-speaker unit for reproduction of sounds in a medium approximating the acoustical properties of flesh, in this case a water-filled plastic bag on which either a bell- or diaphragm-type stethoscope chest piece could be placed for auscultation. Responsiveness of this "artificial precordium" at the various frequencies and amplitudes involved in the experiment was measured by a calibrated direct contact pickup[3] previously described. This intensity calibration was then inscribed on a concealed dial of the attenuator volume control in such a way that dial readings could be noted

by the operator but were not visible to the subject. The subject was asked to place his stethoscope on the transducer, then to turn up the control (several complete rotations were required) until the murmur first became audible. The reading, in decibels on a relative scale, was entered in the data as his auscultatory threshold for the murmur.

Purpose of the second channel was to reproduce background noise in the room through an amplifier and loud speaker at a level comparable to that existing on a busy hospital ward. Actual background noise recorded in another room was used for the test. It likewise was reproduced from a short continuous tape

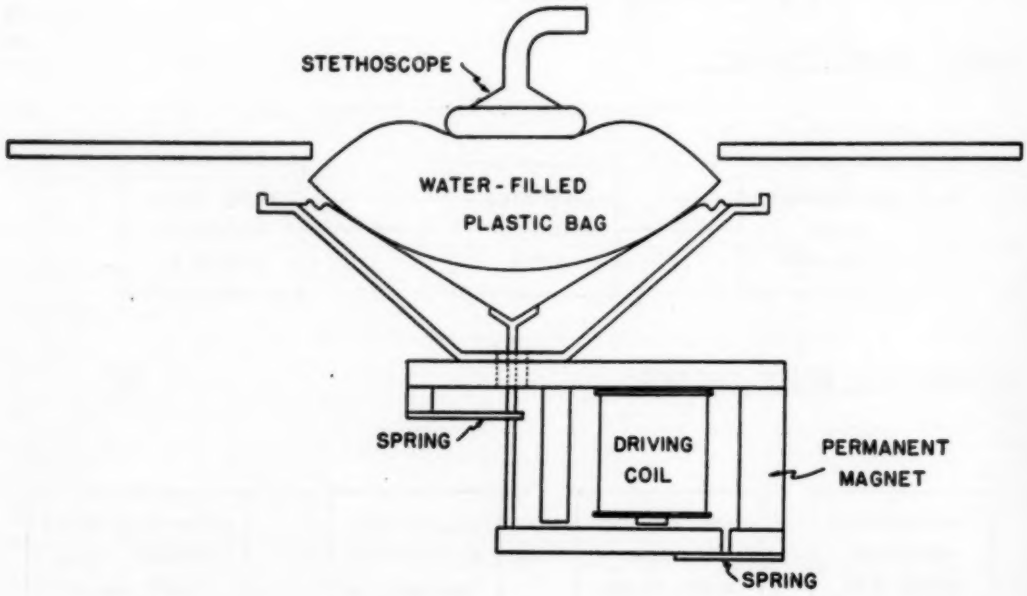


Fig. 2.—Heart sound transducer. The "artificial precordium" designed to permit use of stethoscopes in a manner simulating that of actual auscultation. The electrical potentials of the recorded heart sounds and murmur were transformed by this magnetic transducer into mechanical vibrations in a water-filled medium having the approximate acoustical impedance of flesh.

loop, but in this case the loop was played backward through the machine to render indistinguishable any words or familiar sounds in the noise. This artificial background noise to which the subject was exposed was measured with the sound level meter and held quite constant at a level of 65 db. Total sound pressure within the room under these circumstances was 71 db, an increase of 18 decibels over that measured with the room quiet.

Forty physicians, members of the faculty, the resident and intern staffs of this institution, served as subjects in this experiment. Each was asked to bring with him the stethoscope to which he had become accustomed. First with the room quiet and then with the noise channel running he adjusted the attenuator control, as shown in Fig. 3, to the point at which the murmur was first perceptible through his stethoscope. Readings of three trials were recorded by the operator and averaged to obtain the audibility thresholds charted in Fig. 4. Actually, thresholds for the heart sounds as well as the murmur were recorded but only those for the murmur are illustrated in this report.

The entire procedure was then repeated with the subject listening through a "standard" stethoscope provided for the purpose, an ordinary diaphragm type with plastic tubing. Results obtained when the same stethoscope was used by all subjects are also displayed in Fig. 4.

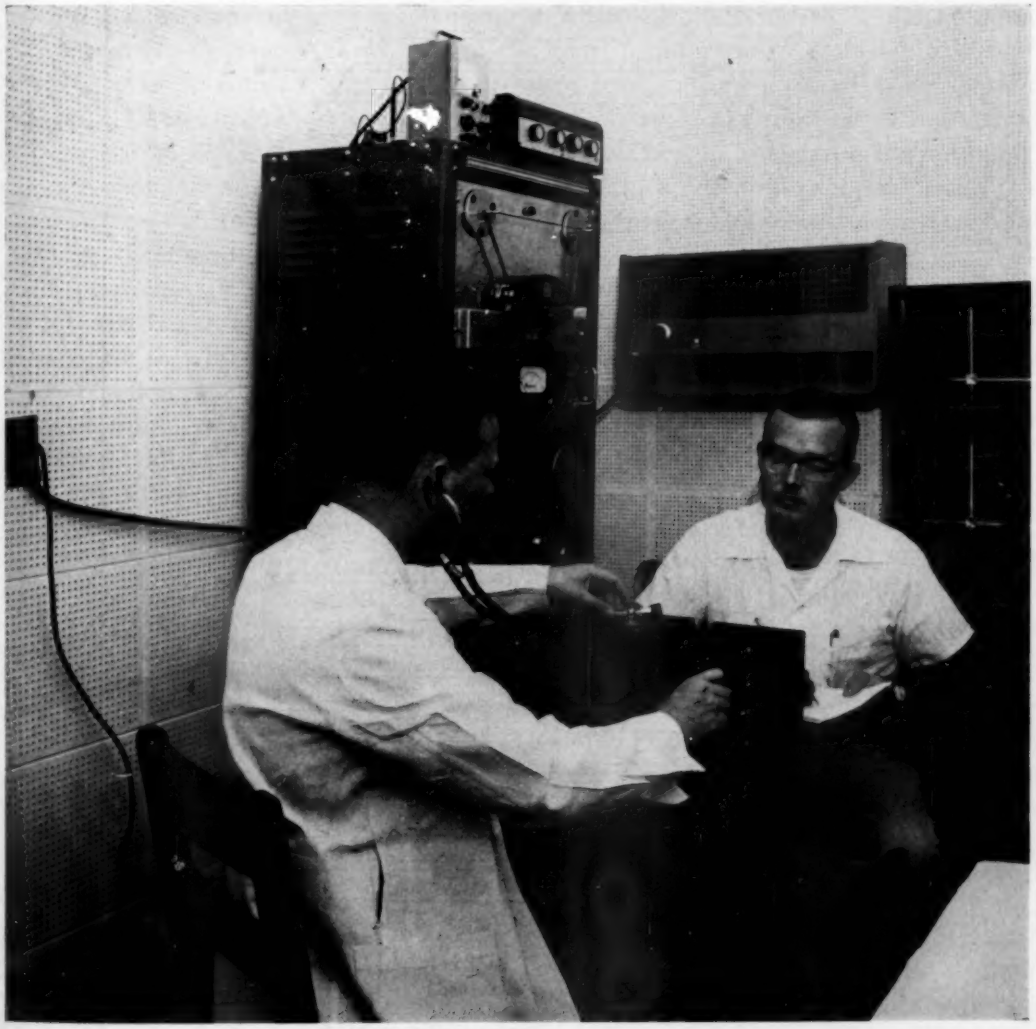


Fig. 3.—Subject listening with stethoscope adjusts volume control until the murmur is first audible. The control setting, visible from behind, is entered in the data by the operator. At left both tape recorder mechanisms with their continuous tape loops may be seen, and at far right is the speaker system for reproduction of background noise. Dimensions of the "sound room" are 6½ by 12 by 10 ft. in height, with inner walls and ceiling built of acoustical tile. Total cost of acoustical remodeling was less than $500.

About 20 to 25 minutes was required for the testing of each subject. The entire experiment was conducted over a period of about a month at the end of which time calibration of the equipment was rechecked and found to have remained unchanged.

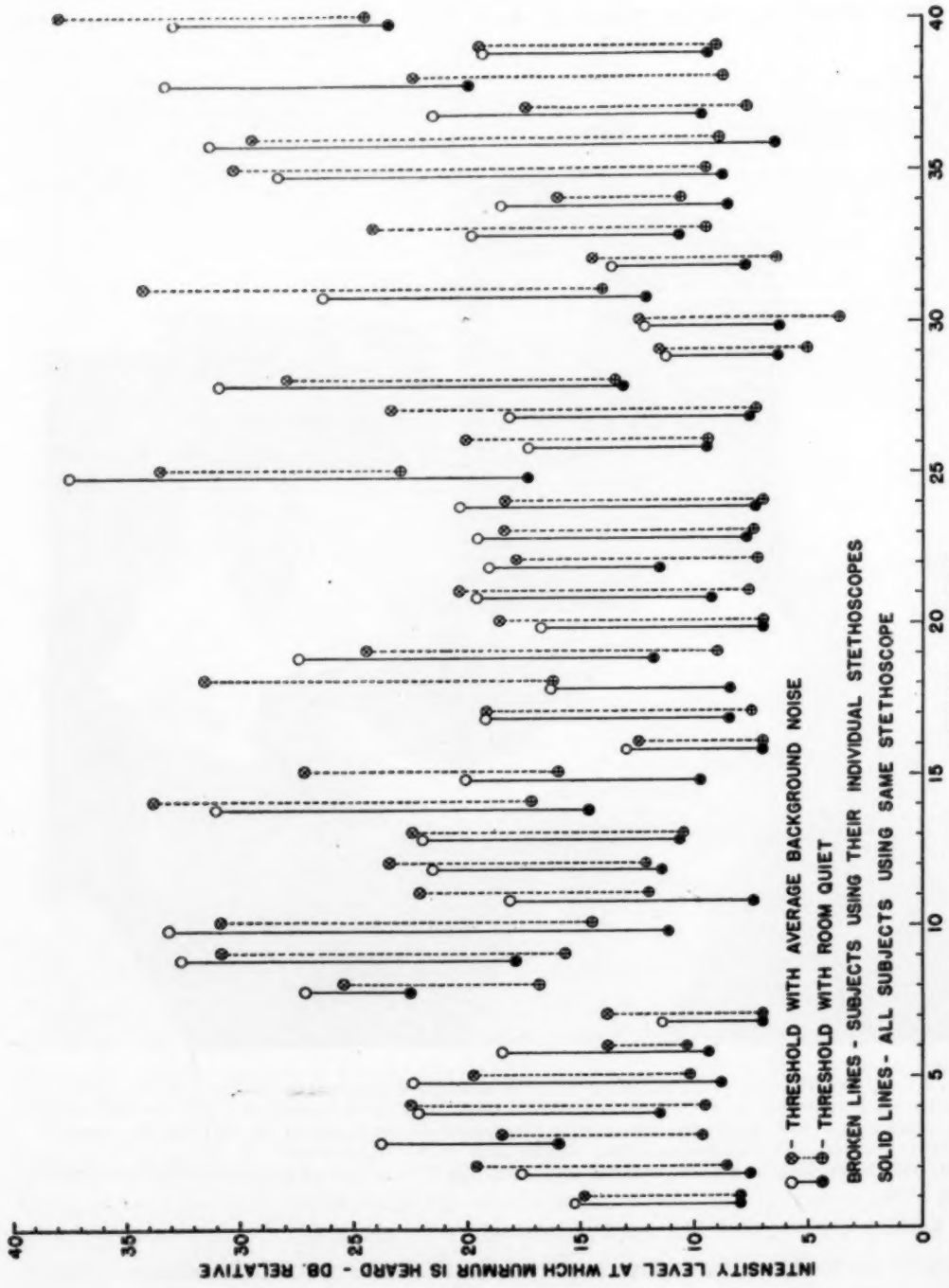


Fig. 4.—Results of experiment showing the effect of background noise on the auscultatory thresholds of forty physicians listening to a heart murmur. Length of the vertical lines indicates the required increase in intensity of the murmur for it to be audible when the environmental noise level was raised from 35 to 65 decibels.

DISCUSSION

Without exception, all subjects required an increase in intensity of the murmur for it to be perceived when the background noise level was raised from that of the quiet room to that of the ordinary examining room. Average increase for the forty subjects was 11.8 decibels when they listened with their individual stethoscopes, or 11.3 decibels when all used the same stethoscope. (Comparable figures for thresholds of the heart sounds were 7.8 and 7.1 decibels, respectively.) Raising the sound pressure level of background noise 18 db, therefore, necessitated an average intensity increase of the murmur of more than 12 times (11 decibels) that required for audibility in the quiet environment. It is reasonable to assume that in a more adequately sound-proofed room—one in which no extraneous sounds were audible—a further improvement in auscultatory proficiency might be realized.

That this difference in audibility threshold is actually due to the noise level difference and not to some learning factor in the experiment is evident from the procedure in which all subjects listened first in the quiet environment and presumably had an opportunity thereby to become more familiar with the sounds. Furthermore, reversing the order of the procedure in several instances yielded similar results.

Doubtless individual differences in hearing acuity, in professional training and experience,* and in stethoscopes employed all influence one's auscultatory ability. These factors were, of course, common to both sets of background noise conditions. Likewise, Fig. 4 shows quite parallel results for the same subject using two different stethoscopes. Actually these individual differences among the 40 subjects produced less over-all variation in thresholds when the noise level was low than when it was high (standard deviation of 4.3 as against 6.8 db), an incidental finding which suggests improved consistency and accuracy of auscultatory performance in the quiet environment.

It is possible that psychologic reactions such as diversion of the subject's attention might also have influenced the results. The same reactions could be expected, however, to similarly alter the threshold in clinical practice as well as under the conditions of this experiment.

The main portals of entry of background noise into the enclosed stethoscopic system would appear to be: through the tubing (primarily the flexible tubing because of its lesser stiffness and mass), sound leaks around the ear pieces or between the chest piece and precordium or in any of the stethoscope parts or fittings, and transmission by the chest wall itself to the area underlying the chest piece. Possibly the listener's bone conduction of extraneous sounds might also play a role. Relative importance of these various factors is the subject of

*In this regard, an analysis of the data in terms of the physicians' clinical experience is noteworthy. Cardiologists (Subjects 5, 25, 28, 31, 35, 36) required the greatest increase in intensity of the murmur when the background noise level was raised, their average being 18.5 db with a range of 14 to 25 db. Pathologists and others in the nonclinical fields (Subjects 26, 29, 32, 34) required the least: 4 to 11 db, average 8 db. The groups of surgeons, obstetricians, pediatricians, and interns all gave results averaging between 10 and 12 decibels. If we grant to the cardiologists the greatest discernment in the matter of heart murmurs it would appear that noise interferes with auscultation even more than results for the entire group would indicate. And it might be expected from a "signal-to-noise ratio" consideration that this change in noise level would produce a more nearly equal change in threshold for the murmur.

tening to a heart murmur. Length of the vertical lines indicates the required increase in intensity of the murmur for it to be audible when the environmental noise level was raised from 35 to 65 decibels.

subsequent experiments. The conclusion remains that commonly encountered levels of background noise can and do seriously impair the physician's ability to hear murmurs through the conventional stethoscope.

Of clinical interest is the fact that, prior to the attempt to measure this impairment reported herein, a surprising degree of increase in one's auditory perception of faint murmurs had been noted in examining patients in the sound-proofed room. It was soon apparent that auscultatory findings which were controversial or questionable on the hospital ward could be more accurately evaluated in the "sound room," and that in numerous instances it was possible to detect murmurs of early valvular disease which were completely inaudible under ordinary conditions of examination inside the room. These clinical observations did, in fact, lead to the present study.

Consideration of the reasons why one's ability to hear should be effectively increased in this way led to some speculation. Doubtless the major reason is that of the improved heart-sound to room-noise ratio with consequent decrease in the masking effect. The "drowning out" or masking of one sound by another, much as even the comparatively loud sounds of conversational speech are masked by the noise of a distant airplane or a radio playing in the room, is a well-known characteristic of human hearing and can be shown to depend in large part upon the relative intensities and frequencies of the two sounds.* Therefore a reduction in intensity of background noise can be expected to produce a comparable reduction in the threshold of audibility for all other sounds. Another explanation—and this may be a minor consideration if pertinent at all—is the possibility that hearing acuity itself may actually increase in a quiet environment, perhaps by action of the tensor tympani or stapedius muscles, in a way akin to that of dark adaptation of vision. The sense of "fullness" or oppression one feels in his ears on entering a sound-proof room conceivably might be a proprioceptive manifestation of this muscle action. At least it has been observed by others that adaptive changes in sensitivity of the ear do take place with exposure to various levels of noise, and that in an anechoic chamber having an extremely low noise level one's own "heart sounds become very noticeable when a person is in it for any length of time."[4] Certainly a sound-proofed room offers no advantage over any other similarly quiet environment in this respect. However, patients in busy hospitals and clinics are not ordinarily examined in a really quiet environment.

Many sources contribute to maintaining a noise level in a room. Our impression in making the measurements was that, except in the case of very large rooms, such as hospital wards, most of the sources were external to the room. Building vibration from heavy air conditioning and other machinery, transmitted through the walls and ventilating air ducts, may introduce remarkably high levels which go unnoticed by the average urban dweller perhaps be-

---

*An octave band analysis (covering the range 20 to 9,600 c.p.s.) of the recorded background noise used in the experiment revealed its peak energy to be concentrated in the 75 to 150 c.p.s. octave, with 75 per cent of the total noise contained in the 20 to 300 c.p.s. range. It might be noted that the latter range also coincides closely with that of the audible frequencies of the heart murmurs, a situation tending to increase masking effects.

cause of their constancy and their predominantly low frequency, and because such noise is unintelligible. Outside street and traffic noises were prominent in some nonairconditioned rooms. The reverberation through corridors of even quite remote sounds, including those of ordinary conversation and activity, appeared to dominate in some of the examining rooms in spite of closed doors. All external noises reaching a room, as well as those arising within, are then reflected repeatedly from hard surface walls, floor, and ceiling to further contribute to the ambient noise level. No unusual sources were found in our noise survey to suggest that the levels of Table I would differ appreciably from those existing under similar circumstances in other institutions.

While these noise levels may seem surprisingly high for hospitals, which traditionally are quiet places, they are doubtless appreciably lower than those encountered in many military induction centers, schools, and industrial areas where large numbers of individuals are examined, often in groups. Murmurs of much less than moderate intensity must certainly pass undetected under such circumstances, regardless of the examiner's hearing acuity or professional ability. Reduction of hospital noise to levels on the order of 35 db ordinarily could not be accomplished economically except in the sound-proofed examining room. The results of this study indicate that such noise reduction is practicable and affords a very real improvement in auscultatory proficiency.

SUMMARY AND CONCLUSIONS

In hospitals and clinics, as in other busy institutions, there is a more or less constant level of background noise. Measurements with a sound-level meter reveal it to be surprisingly high, on the order of 60 to 70 decibels.

Sounds transmitted through conventional stethoscopes are of relatively low intensity and are readily masked by extraneous noise. Particularly is this true of the less obvious murmurs of early valvular disease.

Remodeling a typical examining room with simple sound-proofing measures reduced its background noise to a level of 35 decibels. The effect of noise on the auscultatory performance of forty physicians was then measured under conditions simulating those of actual stethoscopic examination. Average results for the group indicated that the same murmur which could be heard in the quiet room had to be increased to more than 12 times the intensity to be detected under the noise conditions encountered in hospital wards and examining rooms.

It is concluded that, regardless of a physician's hearing acuity or professional experience, ordinary levels of background noise can and do seriously impair his ability to hear heart murmurs. These findings are in agreement with clinical observations indicating that provision of a really quiet examining room affords a worthwhile improvement in auscultatory diagnosis.

Special thanks are due three engineers in the field of acoustics: David C. Apps of the General Motors Noise and Vibration Laboratory, Milford, Mich., and W. A. Munson and R. R. Riesz of the Bell Telephone Laboratories at Whippany and Murray Hill, N. J. Their comments and suggestions on some of the technical aspects of this study have been most helpful.

REFERENCES

1. Peterson, A. P. G., and Baranek, L. L.: Handbook of Noise Measurement, General Radio Company, Cambridge, Mass., 1954.
2. Olson, H. F.: Elements of Acoustical Engineering, ed. 2, New York, 1947, D. Van Nostrand Company, Inc.
3. Groom, D., Underwood, A. F., Bidwell, J. B., and Lindberg, E.: The Recording of Heart Sounds and Vibrations. I. Historical Review and Description of a New Electronic Direct Contact Vibration Pickup, Exper. Med. & Surg. In press.
4. Munson, W. A.: Bell Telephone Research Laboratories, Personal communication—letter dated April 18, 1955.

Clinical Reports

PULMONIC VALVULAR REGURGITATION DURING TWENTY-SEVEN YEARS AFTER GONORRHEAL ENDOCARDITIS

Report of a Case With Catheterization Data

Knud Henning Olesen, M.D., and Jørgen Fabricius, M.D.

Copenhagen, Denmark

ORGANIC pulmonic valvular insufficiency is a rare disorder. The literature on the subject is largely based upon reports of single cases in which the diagnosis was most frequently made at autopsy, more seldom at clinical examination. Good surveys of the literature have been given by Barié (1891),[1] Winkler (1894),[20] Holzmann (1931),[6] and Vellguth (1931).[18]

With the introduction of the technique of cardiac catheterization, an objective verification of a hemodynamically significant pulmonic valvular insufficiency has become possible. The fact that, in spite of the widespread use of this technique, only two cases of pulmonic valvular insufficiency verified by catheterization have been published so far (Joly, Carlotti, and Sicot in 1951[7]; and Kohout and Katz in 1955[8]), must be considered evidence of the rarity of the disease. In view hereof, we consider a report of a case of pulmonic valvular insufficiency with characteristic findings at cardiac catheterization in a 45-year-old woman to be of interest.

CASE REPORT

L.K.P., a 45-year-old woman, was in Medical Department B, Rigshospitalet, University Hospital, Copenhagen, from Feb. 16 to March 14, 1952, and from Aug. 31 to Oct. 12, 1954 (Case Record No. 1102/54). The case report has been supplemented by data available from nine admissions to Danish hospitals during the period from 1927 to 1953.

The patient was of a healthy family. She had been in good health during childhood and adolescence (no rheumatic fever, chorea minor, or syphilis).

At the age of 18 years she contracted gonorrhea, which was complicated by joint affection and a rise of temperature. The joint symptoms subsided in a few weeks, but intermittent pyrexia persisted for almost one year; her general health was highly influenced, she had a medium severe anemia, and she was therefore admitted to Department 2 of Kommunehospitalet, Copenhagen,

---

From Medical Department B, Rigshospitalet, University Hospital, Copenhagen. Head: Professor Erik Warburg, M.D.

Received for publication Dec. 21, 1955.

on Nov. 22, 1927, discharged on June 5, 1928, and to Thisted Hospital on June 5, 1928, discharged on Nov. 25, 1928. During the course of the disease she had prolonged proteinuria with lumbar pain, transitory hematuria, and an elevated blood urea level. Four months after the onset of the disease there occurred an acute aggravation with a stitch in her chest, dyspnea, cough, hempotysis, a rise of temperature, and auscultatory signs of infiltration in the right lung. Clinical examination of the heart, which had shown normal findings at the onset of the disease, revealed a loud diastolic murmur at the left sternal border and distinctly increased cardiac dullness five months later. The electrocardiogram simultaneously showed sinus tachycardia with isoelectric T waves and a normal QRS axis. Ophthalmoscopy about the middle of the period of disease showed a great number of small yellowish white, slightly elevated round plaques, which were considered septic emboli. Examination of cervical secretion showed typical gonococci, and the serum gave a positive, Grade 9 gonococcus complement fixation reaction. Two cultures from the blood were sterile. The patient was treated with salicylates, antipyrine, camphor mixture, staphylococcus vaccine, and septacrol, all without effect. After one year's confinement to bed the patient was afebrile, and slowly recovered.

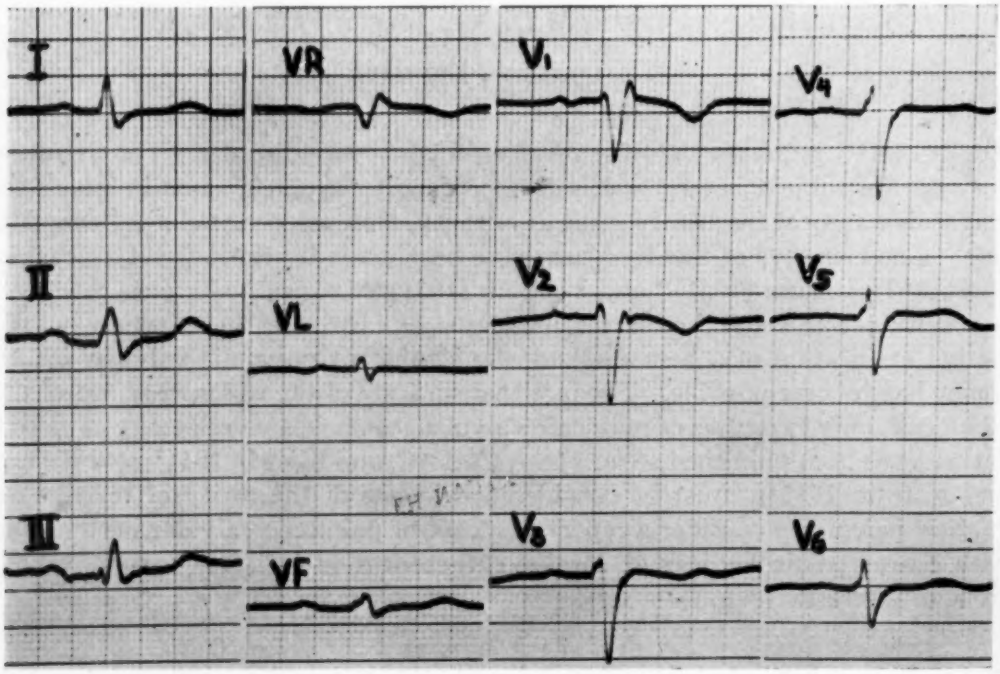


Fig. 1.—Electrocardiogram. Incomplete right bundle branch block. Clockwise rotation.

During the years that followed she was troubled by dyspnea on exertion, palpitation, and premature beats. During a pregnancy at the age of 23 her symptoms increased; she was then examined in the Medical Outpatients Department, Rigshospitalet. A systolic murmur was found at the apex. Radiologic examination showed normal shape and size of the heart, but at fluoroscopy "a very violent pulsation of the pulmonary artery, which dilated enormously at each systole and collapsed completely during the diastole," was noticed (Professor Flemming Møller). The patient carried through her pregnancy, and delivered a living child.

At the age of 26 the patient went through another pregnancy, and gave birth to a living child. Owing to increased heart trouble she was admitted to Obstetric Department A, Rigshospitalet, where delivery was effected by cesarean section and a sterilization operation was done at the same time. Postoperatively she developed pulmonary embolism, but soon recovered from this.

From her twenty-sixth year the patient's dyspnea on exertion and palpitation had slowly increased, and she had been troubled by attacks of oppressive midprecordial pain radiating into both arms, generally persisting for hours or days. At the age of 39 she had had pulmonary embolism and, from her forty-second year, edema of the ankles for periods, for which she was first admitted to this department in 1952. A benign ovarian cyst was removed in 1953 in Gynaecologic Department I, Rigshospitalet. Following this operation she developed pulmonary embolism. Owing to increasing dyspnea and precordial pain, the patient was again admitted to this department in 1954.

She had received disablement pension from her twenty-seventh year. She had been able to perform light domestic work up to her thirty-eighth year; since then she has been practically unable to work, and has been confined to bed for long periods.

Physical Examination at Last Admission.—Examination showed a 45-year-old woman of normal build and in a state of medium nutrition who had no cyanosis or dyspnea at rest and was psychically normal. There was distinct congestion of the cervical veins.

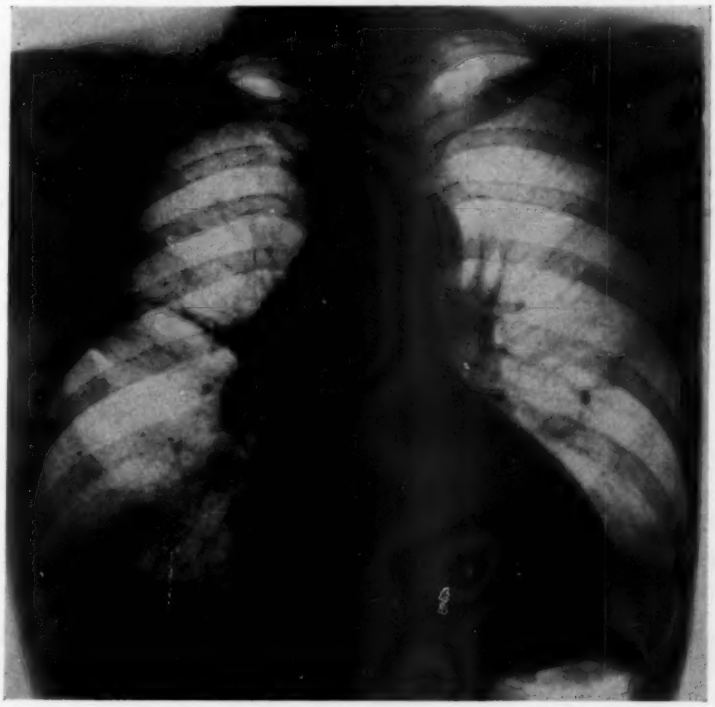


Fig. 2.—Radiograph of the thorax. Heart increased in width. Bulging ascending aorta. Streak-shaped density in right lung field.

Clinical examination of the heart: No precordial bulge or thrill. Vigorous pulsation was visible and palpable downward along the left sternal border and in the upper epigastrium. The impetus was not increased. The apex beat was weak in the fifth intercostal space 1 fingerbreadth laterally to the midclavicular line. A faint, harsh systolic and a faint, blowing diastolic murmur were heard with maxima in the third and fourth intercostal spaces along the left sternal border. These murmurs were distinctly heard in the upper epigastrium, but were only very slight in the second left intercostal space and could not be heard at all in the second right intercostal space. The action of the heart was regular. The second heart sound was faint both on the pulmonic area and on the aorta. Peripheral pulses were normal; there were no signs of aortic incompetence. Auscultation of the lungs: Normal findings. The liver was felt 3 fingerbreadths below the right costal margin; it was not pulsating. There were no ascites or edemas.

Special Investigations.—Height 170 cm. Weight 59.6 kg. Erythrocyte sedimentation rate 6 mm. per hour. Hemoglobin level 101 per cent. Urine: No albumen, blood, pus, or sugar. Microscopy of urine: Normal. Blood urea 25 mg. per 100 ml. Spontaneous specific gravity of urine: 1.008 to 1.024. Blood pressure: 190/110 to 180/110 mm. Hg.

Fig. 3.—Kymogram of the heart in the right anterior oblique position with large pulsations of the pulmonary artery.

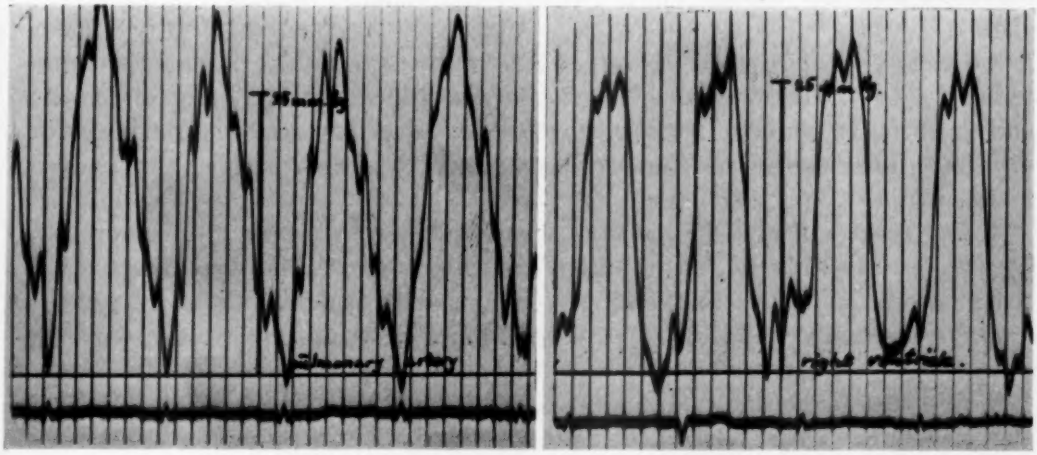
Ophthalmoscopy: Retinal arteries slender, of varying caliber, and with Gunn's phenomena. No hemorrhages or exudates. Wassermann's reaction negative. Gonococcus complement fixation reaction positive of Grade 9 (remained unchanged after intensive penicillin treatment). Secretions from the urethra and the cervix: No gonococci. Electrocardiogram: Incomplete right bundle branch block. Pronounced "clockwise rotation" (Fig. 1).

Radiologic Examination of the Thorax.—The heart was increased in width, mostly to the left, measuring 16 cm. across. Width of the thorax was 30 cm. The ascending aorta showed pronounced bulging to the right, but was not dilated. There was a streak-shaped density in the middle of the right lung field. The vascular markings of the lungs were not increased (Fig. 2). Fluoroscopy and kymography showed very vigorous pulsation of the right ventricle and especially of the pulmonary artery (Fig. 3).

Cardiac Catheterization.—This was first performed in 1952, but the tracings were imperfect; the examination was therefore repeated in 1954. The results of the latter examination may be given here. The catheter passed into the pulmonary artery without difficulty, and a mean pulmonary capillary venous pressure at rest of 5 mm. Hg was measured. The pressure curve from the pulmonary artery showed a systolic pressure of 28 mm. Hg and a diastolic pressure of 0 mm. Hg, so that the pressures were quite identical with those recorded in the right ventricle. This finding could

be repeatedly reproduced (Fig. 4). After raising the patient's legs for three minutes, the pulmonary capillary venous pressure rose to 10 mm. Hg, and the pressure in the pulmonary artery rose to 42 mm. Hg. The mean pressure in the right atrium was normal. (2 mm. Hg). The output was measured as 3.0 L./M.^{2} at rest, and 4.5 L./M.^{2} after exercise. The oxygen saturation in the femoral artery was 94 per cent, in the pulmonary artery 63 per cent.

On the basis of these findings the diagnosis of "organic pulmonic insufficiency" was established.



A. *B.*

Fig. 4.—Curves traced at cardiac catheterization from the pulmonary artery (*A*) and from the right ventricle (*B*). Diastolic pressure 0 mm. Hg in the pulmonary artery.

EPICRISIS

At the age of 18 years, a woman without previously known heart disease developed a gonococcal sepsis of one year's duration. In the course thereof she presented a diastolic murmur along the left sternal border, enlargement of the heart, pulmonary embolism, retinal emboli, and signs of kidney disease. Radiologic examination when the patient was 23 years of age showed "hilar dance." From the age of 18 she had increasing dyspnea on exertion, palpitation, and premature beats; later on, repeated pulmonary embolism, attacks of precordial pain, and tendency to edema. Examination when the patient was 45 revealed faint systolic and diastolic murmurs along the left sternal border, distinct external pulsation corresponding to the site of the right ventricle, radiologic enlargement of the heart with "hilar dance"; cardiac catheterization showed a very low diastolic pressure in the pulmonary artery, identical with that in the right ventricle. On this basis the diagnosis of organic pulmonic valvular insufficiency was made.

COMMENTS

Gonococcal Endocarditis.—The picture of the initial infection with the prolonged septic condition, with development of a diastolic murmur along the left sternal border and enlargement of the heart, and with the occurrence of pulmonary emboli, kidney disease, and retinal emboli is in complete accord with the descriptions of bacterial endocarditis in the right side of the heart given, for instance,

by Langéron and Gardère (1927),[10] Holzmann (1931),[6] Edgren (1937),[5] Lassen (1939),[11] Bartels (1944),[2] and by Lange and Mundt (1954).[9]

The positive gonococcus complement fixation reaction and the demonstration of gonococci in cervical secretion established the diagnosis of gonococcal infection. Two cultures from the blood were sterile, but it is well known in the literature that in cases of gonococcal endocarditis or sepsis it may be difficult to cultivate this organism from the blood (Thayer in 1926[17]; Williams, in 1938[19]; and Davis, in 1940[4]).

Gonococcal endocarditis is rare. Thayer, who took a special interest in this, found twenty-three cases of gonorrheal etiology among 198 cases of bacterial endocarditis (12 per cent). Perry's collective series[13] comprised—after deduction of Thayer's series—fifteen cases of gonococcal endocarditis in 694 of bacterial endocarditis (2.2 per cent).

The valves affected in gonococcâl endocarditis have been best investigated by Thayer. In his series of autopsied cases the aortic valves were affected in 67 per cent, the mitral valves in 19 per cent, the tricuspid valves in 24 per cent, and the pulmonic valves in 24 per cent of the cases. Isolated affection of the pulmonic valves occurred in 19 per cent. Thayer, thus, most frequently found affection of the aortic valves, but stressed that gonococcal endocarditis is the type that shows the highest relative incidence of affection of the pulmonic valves. This view was later supported by Ziegler (1934)[21] and by Perry (1936).[14]

Prior to the era of antibiotics, the course of gonococcal endocarditis was very grave. In Sagot's collective series of 1920,[16] fifty-four out of fifty-eight patients thus died during the acute phase of the disease. In Thayer's series,[17] all twenty-three patients died; in Williams' series,[19] all twelve patients died, and in that reported by Davis[4] there were twelve deaths among sixteen patients. The course may be protracted. In Thayer's series, two-thirds died within two months, whereas the last third had a subacute course of the disease, ending fatally in from two to nine months.

Some of the patients surviving the acute gonococcal endocarditis later developed signs of chronic valvular disease; however, the descriptions of these cases are generally insufficient (Sagot,[16] Newman,[12] Perry,[14] and Ziegler.[21]) Ziegler's patient is of special interest in this connection; he was a man, aged 30, who, ten years after the acute gonococcal endocarditis, presented a clinical picture interpreted as organic pulmonic insufficiency owing to the finding of systolic-diastolic murmurs on the pulmonary area and the demonstration of "hilar dance."

Diagnosis of Organic Pulmonic Valvular Insufficiency.—The clinical diagnosis of this disorder has hitherto been very doubtful; this being due both to the fact that the disease is so rare that the individual clinician cannot attain any high degree of personal experience, and to the fact that the clinical differential diagnosis against aortic valvular incompetence may be very difficult.

The clinical findings on which the diagnosis of pulmonic valvular insufficiency has been based are: (1) A blowing diastolic murmur in the second left intercostal space near the sternum, also audible in the third left intercostal space, with absence of pulsation signs of aortic incompetence; (2) Vigorous pulsation of the right ventricle, visible and palpable downward along the left sternal border

and in the upper epigastrium; (3) Vigorous pulsation of the pulmonary artery, visible at fluoroscopy and kymography ("hilar dance"). To these signs may be added: (4) Enlarged heart with rounded contour and dilatation to the left, which, according to Plesch,[15] is due to an enlarged right ventricle and forward rotation of this. Furthermore, in some cases: (5) Right axis deviation in the electrocardiogram.

In our case, the external pulsation of the right ventricle and the radiologic pulsation of the pulmonary artery were distinctly visible. The shape of the heart at radiologic examination was apparently in accord with the descriptions given in the literature. On the other hand, neither murmurs nor electrocardiographic findings lent definite support to the clinical diagnosis.

A decisive aid to the diagnosis was afforded by the findings at cardiac catheterization. The pressure curves from the pulmonary artery showed a diastolic pressure of 0 mm. Hg, quite identical with that recorded in the right ventricle, so that, hemodynamically, there were no signs at all of closure of the pulmonic valves during the diastole.

We have seen such findings at cardiac catheterization described in only two cases in the literature; one was the case of a patient with congenital interventricular septal defect and pulmonic hypertension (Joly, Carlotti, and Sicot in 1951[7]), the other that of a patient with congenital pulmonic stenosis and insufficiency, and interventricular septal defect (Kohout and Katz in 1955[8]).

Prognostic Considerations.—It appears from the literature that the great majority of cases of pulmonic valvular insufficiency died during the stage of acute or subacute endocarditis. With regard to chronic pulmonic valvular insufficiency, the descriptions of cases are so incomplete that nothing can be concluded with certainty as to the prognosis (Barié,[1] Winkler,[20] Holzmann,[6] Vellguth,[18] and Bourne[3]).

Our patient has lived for 27 years after the acute endocarditis. Her right ventricle thus showed a pronounced capacity for compensating the pulmonic valvular insufficiency; we think this observation is worth mentioning when the question of pulmonic valvulotomy involves the risk of producing pulmonic valvular insufficiency.

SUMMARY

We report the case of a 45-year-old woman, presenting pulmonic valvular regurgitation developed after a gonorrheal endocarditis at the age of 18. The value of heart catheterization as an aid in the diagnosis of pulmonic valvular regurgitation is stressed.

REFERENCES

1. Barié; E.: Recherches sur l'insuffisance des valvules de l'artère pulmonaire, Arch. gén de méd. Paris, Series 7, **28**:30-70, 183-214, 1891.
2. Bartels, E. D.: Endocarditis i højre Hjertehalvdel, Ugesk. f. Laeger **106**:275-82, 1944.
3. Bourne, G.: Pulmonary Regurgitation, Lancet **233**:1427-29, 1937.
4. Davis, J. S., Jr.: Diagnosis and Treatment of Gonorrheal Septicemia and Gonorrheal Endocarditis, Arch. Int. Med. **66**:418-440, 1940.
5. Edgren, W.: Ett fall-av endocarditis pulmonalis med multipla lunginfarkt, Finska läk.-sällsk. handl. **80**:151-62, 1937.
6. Holzmann, M.: Ueber septische Endokarditis der Pulmonalklappen. Ztschr. Klin. Med. **115**:209-243, 1931.

7. Joly, P. Fr., Carlotti, J., and Sicot, J. R.: Les communications interventriculaires (diagnostic par cathétérisme), Arch. mal. coeur **44**:602-624, 1951.
8. Kohout, F. W., and Katz, L. N.,: Pulmonic Valvular Regurgitation, Am. Heart J. **49**:637-42, 1955.
9. Lange, J., and Mundt, E.: Die rechtsseitige Endokarditis des normalentwickelten Herzens, Deutsch. Arch. klin. Med. **201**:476-89, 1954.
10. Langéron, L., and Gardère, H.: les endocardites infectieuses des valvules sigmoïdes de l'artère pulmonaire, J. de méd. de Lyon. **8**:603-609, 1927.
11. Lassen, H. C.: Pneumococendocarditis, Ugesk. f. Laeger **101**:99-104, 1939.
12. Newman, A. B.: The Prognosis in Gonococcal Endocarditis, Am. Heart J. **8**:821-33, 1933.
13. Perry, M. W.: Further Note on a Case of Gonorrheal Endocarditis With Recovery, Am. J. M. Sc. **185**:394-95, 1933.
14. Perry , C. B.: Bacterial Endocarditis, Bristol, 1936, John Wright & Sons, Ltd.
15. Plesch, J.: Herzklappenfehler: In Kraus, F., and Brugsch, T.: Speziellen Pathologie und Therapie innerer Krankheiten. Vol. 4, Berlin, 1925, Urban & Schwarzenberg.
16. Sagot, L.: L'endocardite gonococcique, Thèse de Paris, 1920.
17. Thayer, W. S.: Studies in Bacterial (Infective) Endocarditis, Johns Hopkins Hosp. Reports **22**:1-185, 1926.
18. Vellguth, H.: Zur Pathologie der Pulmonalinsuffizienz, Beitr. path. Anat. allg. Path. **86**:517-42, 1931.
19. Williams, R. H.: Gonococcic Endocarditis, Arch. Int. Med. **61**:26-38, 1938.
20. Winkler, E.: Ueber Insuffizienz der Pulmonalklappen, Inauguraldissertation, Breslau, 1894.
21. Ziegler, K.: Gonorrhoische Endokarditis, München. med. Wchnschr. **80**:2001-2003, 1934.

TRANSIENT VENTRICULAR TACHYCARDIA FOLLOWING THE VALSALVA MANEUVER IN A PATIENT WITH PAROXYSMAL ATRIAL TACHYCARDIA

WILLIAM HOLLANDER, M.D., AND GEORGE ENTWISLE, M.D.

BOSTON, MASS.

THE Valsalva maneuver is occasionally useful in the treatment of paroxysmal tachycardia.[1] To our knoweldge, this is the first case report in which this maneuver appeared to produce transient ventricular tachycardia while it simultaneously relieved the paroxysmal auricular tachycardia. It is of interest that spontaneous reversion of paroxysmal auricular tachycardia to normal sinus rhythm is sometimes accompanied by transient ventricular arrhythmias.[2,3] More common methods used to convert paroxysmal auricular tachycardia, such as carotid sinus stimulation, ocular pressure, Mecholyl, and neostigmine, also at times produce ventricular arrhythmias.[4-6]

CASE REPORT

A 37-year-old white widow was admitted to the Massachusetts Memorial Hospitals because of "pounding pain in the chest" of six hours' duration. The patient was known to have had hypertension for two years, and her chief complaint was migrainelike headaches. Six months prior to admission she had a sudden attack of pounding in the chest, later associated with pressing substernal pain radiating down the left arm. The attack lasted three to five hours and ended after either carotid sinus pressure or ocular pressure was applied.

A similar episode of palpitation without chest pain occurred one month before admission. This attack was recorded electrocardiographically as auricular tachycardia and was terminated by ocular pressure. At this time prophylactic quinidine, 0.2 Gm., three times a day, was begun. Six hours before admission the third episode of pounding in the chest began. Since ocular and carotid sinus stimulation were without effect, hospitalization was advised.

The positive physical findings were confined to the cardiovascular system. Blood pressure was 190/115 mm. Hg, pulse 180, respirations 26, temperature 98.6° F. The heart was enlarged 2 cm. to the left of the midclavicular line in the fifth intercostal space. The rate was rapid and the rhythm regular. A Grade 3 apical systolic murmur was heard. The second pulmonic sound was accentuated. The lungs were clear to auscultation and percussion.

The laboratory data showed a normal urine analysis and hemogram. Electrocardiographic tracing was interpreted as auricular tachycardia.

Hospital Course.—On admission, repeated ocular and carotid sinus stimulation failed to affect the auricular tachycardia. Likewise 0.8 mg. Cedilanid administered intravenously was without apparent immediate effect. Two hours after the administration of Cedilanid, the Valsalva maneuver was employed. During this maneuver, in the phase of forced expiration, the paroxysmal

---

From the Evans Memorial of the Massachusetts Memorial Hospitals and the Department of Medicine, Boston University School of Medicine, Boston.

Received for publication Dec. 21, 1955.

auricular tachycardia terminated, but was unexpectedly replaced by a bout of ventricular tachycardia. Immediately, however, as deep inspiratory breathing began, the unexpected arrhythmia reverted to a normal sinus rhythm which persisted throughout the two-day hospital course.

One month after discharge, the patient was admitted to the surgical service. Transthoracic sympathectomy from D_1 to D_{12} was advised for treatment of hypertension and prevention of future attacks of paroxysmal auricular tachycardia. Since the operation, performed three years ago, the patient has been normotensive and has had no attacks of paroxysmal auricular tachycardia.

DISCUSSION

The Valsalva experiment or maneuver was first described in 1740[7] and is named for its investigator, Antonio Maria Valsalva. The effect of this maneuver on the brachial arterial pressure may be divided into four phases as illustrated in Fig. 1. The first phase occurs immediately after forced expiration begins, the second during the course of sustained forced expiration, the third immediately after the end of forced expiration and the return of deep inspiratory breathing, and the fourth lasts until the blood pressure returns to control levels.

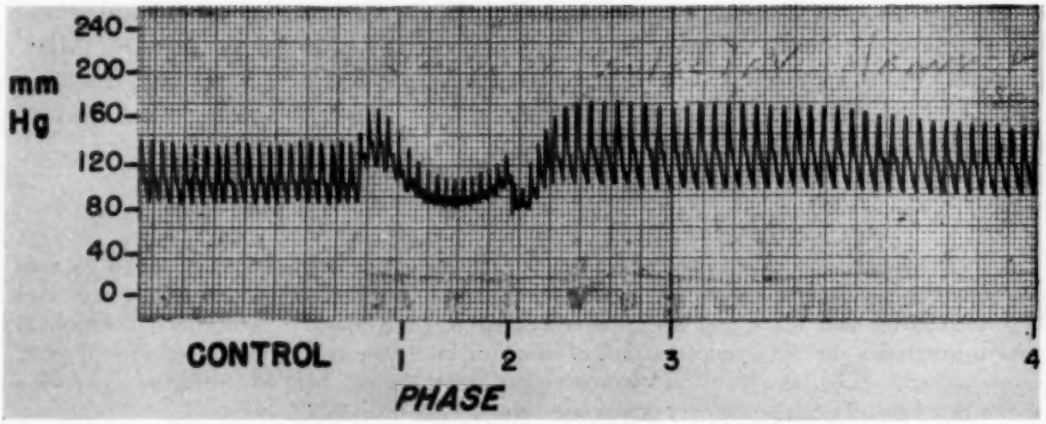


Fig. 1.—Normal brachial artery Valsalva response. See text for details.

During the Valsalva maneuver in normal individuals, premature beats sometimes occur, expecially during the inspiratory effort, Phase 3. The premature beats arising in this phase are attributed to a vagal reflex that occurs secondary to a rise in the systemic blood pressure. This same mechanism may also explain the appearance of premature beats and ventricular tachycardia which have been observed during the conversion of paroxysmal auricular tachycardia.[4-6] The work of Scherf and Chick[8] on the topical effects of acetylcholine on the dog's heart lends support to this hypothesis. The efficacy of Mecholyl (acetyl beta-methylcholine), carotid sinus pressure, ocular pressure, and neosynephrine in the conversion of paroxysmal auricular tachycardia depend on direct cholinergic action, or on reflex vagal stimulation.[17]

Reflex vagal stimulation is also believed to be the mechanism for reversion during or following the Valsalva maneuver in patients with paroxysmal auricular tachycardia, and for this reason the Valsalva maneuver can be grouped with the

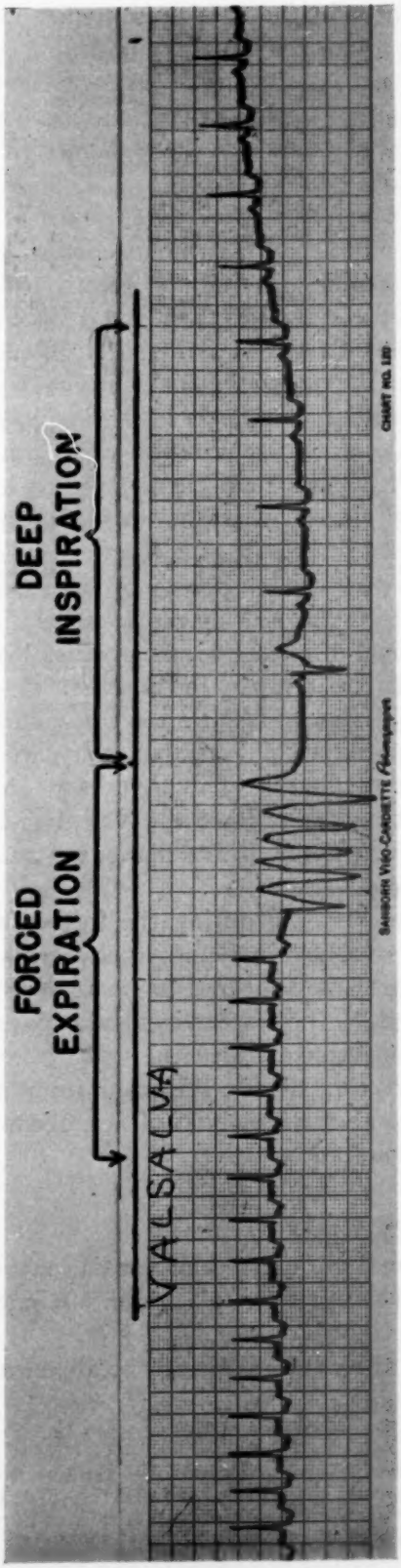


Fig. 2.—Forced expiration versus deep inspiration. ECG tracing shows the appearance of ventricular tachycardia during the Valsalva maneuver in a patient with paroxysmal auricular tachycardia.

above methods of treatment. In the case presented, however, the ventricular arrhythmia definitely took place during the expiratory phase of the Valsalva maneuver. The blood pressure in this patient was not being followed during this maneuver, nor was the patient's expiratory pressure being measured. It is possible that the intrathoracic pressure was low before the onset of the ventricular arrhythmia (Fig. 2) and the arterial pressor response was occurring while the patient was exerting inadequate forced expiration. If this is true, then reflex vagal stimulation can be postulated as the mechanism for both the reversion and the ventricular arrhythmia. During the forced expiratory phase of the Valsalva maneuver, sympathetic rather than vagal discharge probably occurs secondary to a fall in the systemic blood pressure. During this phase, epinephrine is probably released by the sympathetic nervous system and this may produce ventricular tachycardia. This effect of epinephrine appears to require either coexisting myocardial ischemia or reflex vagal inhibition of the higher pacemakers of the heart.[9-12] Myocardial ischemia, during forced expiration when intrathoracic pressure is high, is postulated on the basis of a decrease in coronary blood flow due to an increased resistance to flow and to a fall both in the cardiac output and diastolic blood pressure. Since there is no evidence for inhibition of the higher pacemakers during the blowing effort, the combination of myocardial ischemia and sympathoadrenal discharge seems a plausible mechanism to explain the appearance of ventricular tachycardia in this case.

It is recognized that digitalis, an agent often recommended, and used in this case for the treatment of paroxysmal auricular tachycardia, has been reported to produce ventricular tachycardia.[13,14] The digitalis given to this patient would be having significant effect at the time of the Valsalva maneuver. The close temporal relationship between the Valsalva maneuver and changes in rhythm, however, implicates the maneuver.

The electrocardiographic tracing during the forced expiration (Fig. 2) reveals the dominant rhythm to be ventricular tachycardia for five beats. The ectopic auricular focus at this time appeared to be replaced because (1) it was either completely suppressed by the faster ventricular pacemaker, or (2) it was independently discharging and concealed in the ventricular complexes. The reversion to a normal sinus rhythm during the inspiratory phase of the Valsalva maneuver was likely related to a restoration of neurogenic balance between the cardioaccelerator and cardioinhibitor nerves.

CONCLUSIONS

1. A case of paroxysmal auricular tachycardia, converted during the Valsalva maneuver to a normal sinus rhythm, is presented, in which transient ventricular tachycardia preceded the conversion.

2. The mechanism of the conversion and the appearance of the ventricular arrhythmia have been discussed.

REFERENCES

1. Linenthal, A. J., and Freedberg, A. S.: Measures Used in the Prevention and Treatment of Cardiac Arrhythmias, New England J. Med. **241**:570-578, 1949.
2. Barker, P. S.: Ventricular Tachycardia During an Attack of Paroxysmal Auricular Tachycardia, Heart **11**:67-69, 1924.

3. Hoffman, N. A.: Fibrillation of the Ventricles at the End of an Attack of Paroxysmal Tachycardia in Man, Heart **3**:213-218, 1911-1912.
4. Levine, S. A., and Harvey, W. P.: Clinical Auscultation of the Heart, Philadelphia, 1950, W. B. Saunders Company, p. 327.
5. Meredith, H. C., Jr., and Beckwith, J. R.: Development of Ventricular Tachycardia Following Carotid Sinus Stimulation in Paroxysmal Supraventricular Tachycardia, AM. HEART J. **39**:604-606, 1950.
6. Landman, M. E., and Ehrenfeld, I.: Ventricular Fibrillation Following Eyeball Pressure in a Case of Paroxysmal Supraventricular Tachycardia, AM. HEART J. **43**:791, 1952.
7. Luisada, Aldo A.: Heart, Baltimore, 1948, Williams & Wilkins Company, 653 pp.
8. Scherf, D., and Chick, F. B.: Abnormal Cardiac Rhythms Caused by Acetylcholine, Circulation **3**:764-769, 1951.
9. Wilburne, M., Surtshin, A., Rodbard, S., and Katz, L. N.: Inhibition of Paroxysmal Ventricular Tachycardia by Atropine, AM. HEART J. **34**:860-870, 1947.
10. Hoff, H. E., and Nahum, L. H.: The Role of Adrenaline in the Production of Ventricular Rhythms and Their Suppression by Acetyl-B-Methylcholine Chloride, J. Pharmacol. & Exper. Therap. **52**:235-245, 1934.
11. Lenel, R., Vanloo, A., Rodbard, S., and Katz, L. N.: Factors Involved in the Production of Paroxysmal Ventricular Tachycardia Induced by Epinephrine, Am. J. Physiol. **153**:553-557, 1948.
12. Nathanson, M. H., and Miller, H.: The Action of Norepinephrine, Epinephrine, and Isopropyl Norepinephrine on the Rhythmic Function of the Heart, Circulation **6**:238-244, 1952.
13. Marvin, H. M.: Paroxysmal Ventricular Tachycardia With Alternating Complexes Due to Digitalis Intoxication, AM. HEART J. **4**:21, 1928.
14. Lundy, C. J., and McLellan, L. L.: Paroxysmal Ventricular Tachycardia: An Etiological Study With Special Reference to the Type, Ann. Int. Med. **7**:812-836, 1934.
15. Starr, I., Jr.: Acetyl-B-Methylcholin, Further Studies of Its Action in Paroxysmal Tachycardia and in Certain Other Disturbances of Cardiac Rhythm, Am. J. M. Sc. **191**:210-225, 1936.
16. Donegan, C. K., and Townsend, C. V.: Phenylephrine Hydrochloride in Paroxysmal Supraventricular Tachycardia, J. A. M. A. **157**:716-718, 1955.
17. Kennamer, R., and Prinzmetal, M.: The Cardiac Arrhythmias, New England J. Med. **250**:509-520, 1954.

Book Reviews

ANGIOCARDIOGRAPHIC INTERPRETATION IN CONGENITAL HEART DISEASE. By Herbert L. Abrams, M.D., and Henry S. Kaplan, M.D., Springfield, Ill., 1956, Charles C Thomas, Publisher.

This monograph is devoted to a review of the diagnostic experiences which were recorded in a series of over 350 angiocardiographic and aortographic studies in congenital heart disease. The majority of the studies were performed on infants and children under the age of three years, although in a few instances, older children and adults were studied in this group. A general introductory section includes a discussion of methods of technique, the selection of opaque media, reactions to the procedure, including the possibility of angiocardiographic death, and other types of reaction. The normal sequence of events noted during opacification of the cardiac chambers is also reviewed, including the anatomic relationships presented in various views.

The second section deals primarily with a review of the angiocardiographic findings in specific anomalies, including such common anomalies as tetralogy of Fallot, pulmonary stenosis, and septal defects, as well as the rarer congenital lesions. There are numerous illustrations with good reproductions of the angiocardiograms obtained. These are supplemented by diagrammatic shaded drawings which illustrate the passage of the opaque medium through the various cardiac chambers. These diagrams aid considerably in the understanding and interpretation of the shadows seen in the roentgenograms.

There is a wealth of good diagnostic information available in this monograph, and it will be particularly helpful to those interested in the field of congenital heart disease in children. The reviewer personally wonders, however, whether newer developments in cineangiocardiography, with the use of high speed cameras, may not in time render obsolete some of the present technical methods utilized in this study. Nevertheless, the basic appearance of the contrast shadows presented should still be helpful in understanding visualization of the cardiac chambers in health and disease.

H. E. H.

CARDIOVASCULAR INNERVATION. By G. A. G. Mitchell, Edinburgh and London, 1956, E. & S. Livingstone, Ltd., 356 pages, 202 figures.

In spite of much research on innervation of the heart and vessels no specific monograph on this subject has been available so far. Mitchell's book, based on large experimental experience, fills this gap admirably. Peripheral autonomic innervation as well as central representation are discussed in thorough detail, and the text is supported by many excellent illustrations. Although the study of the anatomic structure is the basis, functional, pathologic, and clinical considerations are discussed and, therefore, the book may be defined as a treatise on applied anatomy. That the material is based essentially on man and, in experimental material, not lower in the scale than the monkey, makes the book particularly valuable for clinical application. The titles of the chapters, in chronologic order, are: I, General Information; II, Autonomic Representation in the Cerebrum; III, Autonomic Representation in the Cerebellum, Brain Stem, and Cord; IV, Autonomic Outflows and Endings; V, Autonomic Afferents and Endings; VI, The Peripheral Parts of the Autonomic System; VII, Innervation of the Vessels in the Head and Neck; VIII, Innervation of Vascular Structures in the Thorax; IX, Innervation of Vessels in Abdomen and Pelvis; and X, Innervation of Vessels in the Limbs. The bibliography extends over 28 pages. The book will be invaluable as a reference source for the internist, surgeon, physiologist, and, last but not least, for the anatomist.

E. S.

MANAGEMENT OF STROKES. By Keith W. Sheldon, Philadelphia and Montreal, 1956, J. B. Lippincott Company, 13 chapters, 134 pages, and 52 figures.

This short book provides an approach to the diagnosis and management of patients with "catastrophic cerebral dysfunction." The author deplores the use of the term "stroke" to include all such cases and very properly emphasizes that traumatic, metabolic, and inflammatory, as well as vascular and neoplastic causes must be considered in the differential diagnosis.

The material is presented in a practical fashion. A number of "do's and don'ts" are listed and analogies, caricatures, diagrams, italics, and brief case histories are used to illustrate important points. The discussion is brief throughout all the chapters but by using a somewhat arbitrary manner of presentation the author has been able to include a surprising amount of information.

The physician who rarely sees patients with sudden, severe loss of cerebral function may find this book to be a useful guide. It will be of little value to the consultant.

J. W. E.

VERHANDLUNGEN DER DEUTSCHEN GESELLSCHAFT FÜR KREISLAUFFORSCHUNG. Edited by Professor Dr. Rudolf Thauer, Darmstadt, 1955, Dr. Dietrich Steinkopff Verlag, 484 pages, 183 illustrations.

As in the previous meetings of the German Society for the Study of Circulation, the general standard of the communications is high. The first day of the meeting was devoted to coronary thrombosis (13 papers, pp. 1 to 162), the second day to cor pulmonale (15 papers, pp. 163 to 384), and the third day to miscellaneous presentations (13 papers, pp. 385 to 463). An appreciable number of guest lecturers (D. E. Gregg, Washington, D. C.; E. A. Nikkilä, Helsingfors; L. Pescador and others, Madrid; J. Gibert-Queralto, and others, Barcelona; G. Daddi, Milan; D. Halmagyi, Szeged; and H. Denolin, Brussels) give the meeting an international flavor.

It is, of course, impossible to review adequately in a condensed space the numerous excellent presentations, or even to give a complete list of the titles (pp. V to IX). A few arbitrary selections may suffice to show some of the central topics.

In the papers on coronary thrombosis, the emphasis is placed on the causes (general and immediate) rather than on the resulting myocardial infarction. The roles of blood lipids, blood coagulation, and anticoagulant therapy are in the foreground of discussion. Nikkilä holds that cholesterol in the beta lipoproteins, rather than the total blood cholesterol, is the most important fraction for the development of atheroma; in 20 to 30 per cent of patients with coronary disease the total blood cholesterol is below the normal average. Hauss found a significantly higher blood content of beta-lipoproteins and neutral fat in patients forty days after a myocardial infarction as compared to normal subjects, but no significant increase in blood cholesterol. On the other hand, Schettler and associates (p. 124) attached significance to an increase of total cholesterol as well as of beta-lipoproteins. Pescador and associates go so far as to predict imminent infarction from the ratio of beta-globulin, gamma-globulin, and cholesterol to heparin.

While anticoagulant therapy is generally considered to be the most important advance in the treatment of myocardial infarction, there are several shades of opinion about the success to be expected. Bansi and associates found macroscopic thrombosis in only 48 per cent of 329 infarct cases. Anticoagulants are contraindicated in the 11 per cent of infarct cases with intramural hematoma; unfortunately, it is impossible to differentiate these cases clinically (Schoen, p. 38). Aschenbrenner (p. 143) stresses the importance of treatment with strophanthin or digitalis preparations in addition to anticoagulant therapy. While, of all causes, thrombus formation is the most important for the development of myocardial infarction, nervous strain, local hypoxia, and sensitization processes are contributory factors according to Jürgens (p. 94). Particularly, the role of local edema is stressed (Jürgens and E. Müller, p. 3) and this is, according to Müller, the major contributory factor in young infarct patients. This is interesting because differences in the pathogenesis of infarction in younger and older patients have been suggested for some time. Thrombosis may be a secondary development to coronary insufficiency, and after hard exercise myocardial infarction may develop with only minor coronary arteriosclerotic degeneration. R. Schoen (p. 38) in his paper on clinical diagnosis and therapy of coronary thrombosis discusses

ECG findings as the most important diagnostic method in somewhat greater detail. One case with initial inversion, preceding the S-T elevation, is demonstrated. This early phase, which has been shown in animal experiments, is rarely seen in man due to the time interval between occurrence of infarct and the first ECG. D. E. Gregg reviews in an excellent paper some hemodynamic aspects of coronary circulation, particularly the role of anastomoses and various surgical procedures designed to stimulate the development of collateral circulation.

Hemodynamic aspects in chronic cor pulmonale are reviewed by R. Knebel (p. 181) and W. Bolt (p. 196), and the pathologic anatomy by E. Kirch (p. 164). N. Denolin's review of "chronic pulmonale in internal medicine" (in French), pp. 217 to 279, including 15 tables, 17 illustrations, and 353 references, is an outstanding contribution to this important problem.

E. S.

Announcements

A COURSE IN PRACTICAL ELECTROCARDIOLOGY will be presented December 3 to 7, 1956, in Houston, Tex., by Dr. Demetrio Sodi-Pallares, Chief of the Department of Electrocardiology at the National Institute of Cardiology, Mexico City, under auspices of the University of Texas Postgraduate School of Medicine and Baylor University College of Medicine.

In addition to Dr. Sodi-Pallares' evening course and his clinical pathologic conference discussions, individual instruction in reading electrocardiograms will be provided by faculty members of the sponsoring institutions. For more advanced students, panel discussions will be held on subjects of special interest to electrocardiologists. Inquiries should be addressed to The University of Texas Postgraduate School of Medicine, Texas Medical Center, Houston 25, Tex.

THE NINTH INTERNATIONAL CONGRESS ON RHEUMATIC DISEASES will be held at Toronto, Ontario, Canada, from June 23 to 28, 1957. This quadrennial function of La Ligue Internationale contre le Rhumatisme will be held under the auspices of the Canadian Rheumatism Association.

The Program Committee invites contributions to the scientific program of the Congress and is anxious to receive reports on current clinical or basic research dealing with any aspect of the rheumatic diseases.

Those offering papers for consideration should submit a 200 to 300 word abstract not later than the first of January, 1957. Abstracts should be submitted in triplicate in the language in which the paper is to be read. If an abstract is submitted in a language other than English, it will be helpful, though not essential, if it can be accompanied by an English translation.

All correspondence should be directed to: The Ninth International Congress on Rheumatic Diseases, Post Office Box 237, Terminal "A", Toronto, Ontario, Canada.